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SEVENTH EDITION

BASIC OBSTETRICS

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PREFACE TO THE SEVENTH EDITION

Writing the preface to a subsequent edition of a textbook is a distinctive privilege. We are pleased that the audience accepted the previous six editions well enough to allow the seventh edition to be published.

Basic Obstetrics continues to improve in this new edition by keeping abreast of new information and updating where appropriate. Theoretical and controversial aspects are minimized. The intent of the book remains the same, which is to provide the basics of the speciality in a well-organized form to students, residents or practitioners.

I am most grateful to the publisher Mr. Sayed Mahmoud for his encouragement and courtesy. Special thanks are due to Doctor Essam Hoballah for his untiring efforts and great skill in typing the manuscript. I also thank Mr. Sayed Abd El-Naby for preparing the illustrations. My appreciation also goes to my colleagues at the Department of Obstetrics and Gynaecology, for the way we work together as a team, and enjoy each other's company as friends. I am also grateful to the staff of Al-Ahram Commercial Press for their efforts to prepare the book.

Farouk Haseeb

Cairo, 2003

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To my family...
Mai, Alaa
and Nevine

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NORMAL PREGNANCY

PHYSIOLOGY OF CONCEPTION

FERTILIZATION

It is the union of ovum (0.1 mm.) and spermatozoon. It occurs at the ampulla of the fallopian tube. After intercourse, the spermatozoa ascend and reach the tubes within 30–40 minutes, but they are not able to fertilize the ovum immediately, and remain 2–6 hours during which they undergo certain enzymatic change called capacitation after which they are able to fertilize the ovum. Capacitation means the activation and release of hydrolytic enzymes by the spermatozoa. These enzymes are necessary for penetration of the zona pellucida of the ovum. The cervical and tubal secretions are mainly responsible for capacitation. The fertilized ovum is called the zygote. The sperms move at a rate of 6 mm/minute. The spermatozoa retain the power to fertilize the ovum for about 2 days but can remain motile in the genital tract for several days.

EARLY DEVELOPMENT

Few hours after fertilization, the zygote divides repeatedly to form a round mass of cells (blastomeres) called the morula. The cells of the morula become grouped into an inner cell mass made of small cells; and a peripheral layer made of large cells. The morula reaches the uterine cavity 3 or 4 days after ovulation, and this is caused by tubal contractions and movements of cilia. Fluid then collects between the inner cell mass and the peripheral layer of the morula to form the blastocyst. The inner cell mass is called the embryonic mass and will form the embryo, while the peripheral layer is called the trophoblast which is responsible for nutrition of the embryo. The inner cell mass remains attached to the trophoblast at one point. The blastocyst remains free in the uterine cavity for 3 or 4 days, during which it is nourished by the secretion of endometrium (uterine milk). About 6 or 8 days after ovulation, the blastocyst becomes embedded into the endometrium.

THE DECIDUA

Immediately after embedding the endometrium is changed into the decidua of pregnancy, i.e. the stroma cells become large with small nuclei and clear cytoplasm and lie closely packed together. The part of decidua lying deep to the embedded ovum and separating it from the uterine muscle is called the decidua basalis. The part of decidua which covers the ovum and separates it from the uterine cavity is the decidua capsularis. The rest of the decidua lining the uterine wall is the decidua parietalis or vera. The cavity

between the decidua capsularis and decidua vera is called the decidual space. As the ovum enlarges and fills the uterine cavity, the decidua capsularis comes in contact and fuses with the decidua parietalis thus obliterating the decidual space. This occurs at the 12th week of pregnancy.

FUNCTIONS OF THE DECIDUA

(1) It is the site of implantation of the fertilized ovum; (2) Protection of the uterine wall against invasion by the chorionic villi; (3) Nutrition of the embryo in the early stage of development as the decidua cells contain fat and glycogen; (4) Formation of the placenta.

FORMATION OF THE CHORION

After implantation of the ovum, the trophoblast becomes differentiated into 2 layers, an outer one which is a sheet of protoplasm without cell margins called syncytium and an inner layer made of round cells called Langhans' layer or cytotrophoblast. A third layer or mesoderm which is primitive connective tissue appears inner to Langhans' layer. The three layers form the chorion. Spaces appear in the syncytium which increase in size and fuse together to form the chorio-decidual space. Erosion of the maternal blood vessels by the trophoblast allows blood to flow in this space.

CHORIONIC VILLI

Appear about the 14th day after ovulation. Primary villi are strands made of syncytium and cytotrophoblast. When they become invaded by mesoderm they form secondary villi. When blood vessels appear in the mesoderm they form tertiary villi. At the 12th week, the villi related to the decidua capsularis disappear leaving a smooth membrane called the chorion laeve while the villi related to the decidua basalis grow and form the chorion frondosum which will form the placenta.

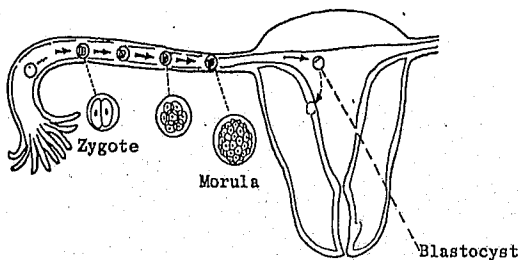


Fig. 1. Fertilization and implantation

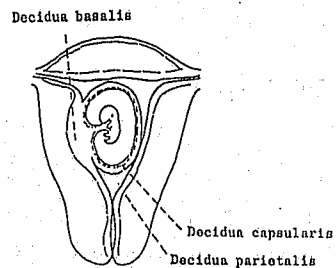


Fig. 2. Decidua

THE PLACENTA

ANATOMY OF THE PLACENTA

At full term the placenta is an oval or circular cake-like organ measuring 15–20 cm. in diameter, about 2.5 cm. in thickness and 500 grams in weight. The fetal surface is covered with the amnion which is a thin smooth membrane. The umbilical cord is inserted in the fetal surface of the placenta usually near its center. Under the amnion, there is the chorionic plate which is few millimeters in thickness and from which arise the villi of the placenta. It is composed of connective tissue and contains branches of umbilical vessels which divide to enter the villi. The maternal surface is dull red in colour and is divided by grooves into 15–20 irregular lobes called maternal cotyledons. The maternal surface is partly covered by a thin grey layer of decidua called the basal plate.

THE PLACENTAL BARRIER

Normally the fetal blood in the chorionic villi is separated from maternal blood in the intervillous spaces by the placental membrane or barrier which is made of syncytium, Langhans' layer, connective tissue stroma of the villous and the wall of the fetal blood vessels. From the fifth month (20th week) the Langhans' layer starts to disappear and from the sixth month the syncytium becomes progressively thinned and so the placental barrier diminishes in thickness.

FUNCTIONS OF THE PLACENTA

- 1) *Nutritive*: Glucose, amino acids and vitamins pass by active diffusion helped by enzymes and not by simple diffusion.
- 2) *Respiratory*: Placenta acts as a fetal lung. Oxygen and carbon dioxide pass by simple diffusion.
- 3) *Excretory*: Placenta acts as a fetal kidney excreting waste products as urea into maternal blood.
- 4) *Hormonal*: All placental hormones are secreted by the syncytium and include the following:
 - a) Steroid hormones: Oestrogens and progesterone. Oestrogens include oestriol (90%), oestradiol, oestrone, and oestetrol (E4).
 - b) Hypothalamic-like hormones: Gonadotrophin-releasing hormone, corticotrophin-releasing hormone, and thyrotrophin-releasing hormone.
 - c) Pituitary-like hormones: Human chorionic gonadotrophin, chorionic corticotrophin, chorionic thyrotrophin, melanocyte-stimulating hormone, prolactin, and human placental lactogen.
 - d) Relaxin.
 - e) Inhibin and activin.

- 5) *Production of enzymes*: As histaminase, oxytocinase and insulinase.
- 6) *Haemopoietic*: It forms fetal haemoglobin.
- 7) *Barrier action*: The placenta can prevent the passage of many undesirable substances as toxins of diphtheria and tetanus and organisms of gonorrhoea and leprosy.
- 8) *Production of proteins* as pregnancy associated plasma protein A, B and C. PAPP-A is increased in pre-eclampsia, and decreased in Down syndrome.

Notes:

- *Human chorionic gonadotrophin (HCG)*: The hormone prolongs the life of the corpus luteum and stimulates it to secrete oestrogen and progesterone to maintain pregnancy till the placenta is formed (luteotrophic effect). It also stimulates the synthesis of steroid hormones by the placenta, and has immunosuppressive activity. It is a glycoprotein and consists of alpha and beta amino acid chains. The alpha subunit is similar to that of follicle stimulating hormone, luteinizing hormone and thyroid stimulating hormone while the beta subunit is specific for HCG. The hormone appears in the maternal blood on the day of implantation and the concentration increases rapidly to reach a peak about the 10th week of pregnancy (about 50,000 mIU/ml) and then it falls to reach a low level at about 18th week (about 5,000 mIU/ml) and remains as such till term. HCG disappears from blood and urine 1 to 2 weeks after delivery and 2 to 8 weeks after abortion and 8–12 weeks after evacuation of hydatidiform mole.
- *Human placental lactogen*: It is luteotrophic, somatotrophic, mammatrophic and lactogenic. It is similar in structure and function to the pituitary growth hormone.
- *mIU/ml* = milli international unit per millilitre.
- *Relaxin*: It is a peptide hormone secreted by the corpus luteum (main source), chorion (placenta), and decidua. Its role during human pregnancy is unknown. It may cause uterine relaxation, softening and effacement of the cervix, and separation of the symphysis pubis during delivery.
- *Placental inhibin* is believed to inhibit the secretion of gonadotrophin-releasing hormone and HCG while *activin* stimulates the release of these hormones.

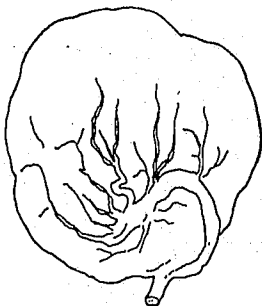


Fig. 3. Fetal surface of the placenta

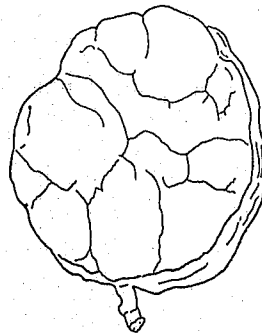


Fig. 4. Maternal surface of the placenta

CONGENITAL ABNORMALITIES OF THE PLACENTA

A) Abnormalities of Shape and Size

1. *Bilobate placenta*: The placenta is made of two equal lobes connected together by a bridge of placental tissue.
2. *Bipartite placenta*: It is made of two equal parts connected together by membranes. There is a single cord which is inserted in one part, or in the membranes between the two parts or it may divide to supply each part.
3. *Placenta succenturiata*: The placenta is made of two unequal parts connected by membranes. The cord is inserted in the big part and branches of the umbilical vessels (an artery and a vein) pass through the membranes to reach the small accessory lobe. This accessory lobe may be retained in the uterus after delivery leading to postpartum haemorrhage or puerperal sepsis. A retained lobe is known when a circular gap is seen in the membranes from which blood vessels pass towards the main placenta. More than one accessory lobe may be present.
4. *Diffuse or membranous placenta*: A great part of the chorion develops into placental tissue. The placenta is large and thin and may measure 15–20 inches in diameter. Because the placenta is large it may reach the lower uterine segment causing placenta praevia.
5. *Circumvallate placenta*: There is a white ring of decidua around the edge of the placenta on its fetal surface. It is about 1 cm in width. It may lead to abortion, preterm labour, intrauterine growth restriction, intrauterine death, abruptio placentae, and slight increase of congenital malformation. In circumvallate placenta, the chorionic villi continue to grow beyond the chorionic plate (placenta extrachorialis).
6. *Fenestrate placenta*: Very rare. The central portion of the placenta is missing.

B) Abnormal Adhesion of the Placenta or Placenta Accreta

The decidua basalis is absent or poorly formed and so the chorionic villi penetrate deeply into the uterine wall. When the villi come just in contact with the muscle layer it is called placenta accreta. When the villi penetrate into the muscle layer it is placenta increta and when they reach the peritoneal coat it is placenta percreta. The condition leads to retention of the placenta after delivery of the fetus.

C) Abnormal Weight

Normally, the placenta at term is about one-sixth of fetal weight. In congenital syphilis, diabetes mellitus and hydrops fetalis the size and weight of placenta increase to be $\frac{1}{3}$ or $\frac{1}{2}$ of fetal weight.

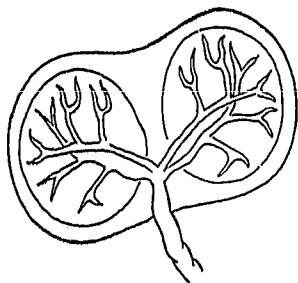


Fig.5. Bipartite placenta

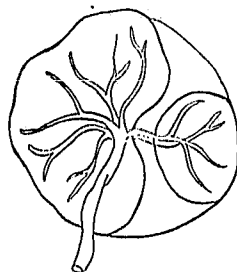


Fig. 6. Placenta succenturiata

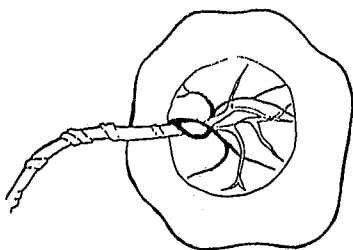


Fig. 7. Circumvallate placenta

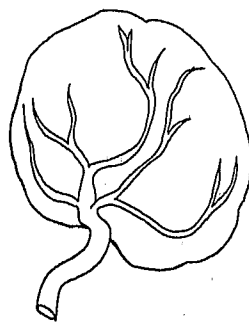


Fig. 8. Battledore placenta

D) Abnormal Position or Placenta Praevia

E) Abnormalities in Insertion of the Cord

The insertion of the cord may be central, eccentric (70%), marginal (battledore placenta) or velamentous. Velamentous or membranous insertion means that the cord is inserted in the membranes outside the placental margin. The umbilical vessels pass through the membranes to reach the placenta. If these vessels pass over the region of the internal Os the condition is called vasa praevia, and rupture of the membranes during labour will lead to antepartum haemorrhage of fetal origin.

THE AMNION

The amnion is a transparent greyish membrane consisting of five layers: an epithelium (cuboidal cells); basement membrane; a compact layer (reticular fibres arranged in bundles); a fibroblastic layer (consisting of fibroblasts and Hofbauer cells) and a spongy layer which contains mucus that allows movement of the amnion upon the chorion. The amnion is devoid of blood vessels, lymphatics and nerves.

CHARACTERS OF AMNIOTIC FLUID

- 1) *Physical properties:* It is colourless, pale yellow or milky from emulsification of vernix caseosa. Specific gravity 1010–1020. Reaction neutral or slightly alkaline (pH 7–7.5). It reaches its maximum volume at 38 weeks (about 1 litre) and gradually diminishes to be 500–1000 ml at term. It is completely changed every three hours.
- 2) *Chemical composition:* Water: 98–99%, solids: 1–2%, half organic and half inorganic. Organic constituents include carbohydrates as glucose and fructose, proteins as albumin and globulins, lipids, hormones as oestrogens and progesterone and enzymes as alkaline phosphatase. The inorganic constituents are similar to those found in the maternal plasma as sodium and chlorides.

ORIGIN OF LIQUOR AMNII

The amniotic fluid has both fetal and maternal origin. It is mainly of fetal origin.

- *Fetal origin:* (1) fetal urine; (2) secretion from the amniotic epithelium; (3) secretion from bronchial mucosa, buccal mucosa and salivary glands; (4) diffusion from the umbilical cord vessels; (5) transudation through fetal skin.
- *Maternal origin:* The liquor is a filtrate from maternal plasma.

Note: The amount of fetal urine ranges from 400 to 1200 ml/day.

FUNCTIONS OF THE LIQUOR AMNII

A) During Pregnancy

1. Protection of the fetus against external trauma.
2. It keeps the fetal temperature constant.
3. It allows free fetal movements thus helping the development of fetal muscles.
4. Prevents adhesions between the amnion and fetal skin.
5. Nutrition as some of the fluid is swallowed by the fetus.
6. Acts as a medium for fetal excretion.
7. Forms a closed sac around the fetus preventing ascent of infection from the cervix or vagina.

B) During Labour

1. The bag of forewaters helps dilatation of the cervix.
2. It prevents direct compression of the placenta between the uterine wall and fetus during uterine contraction thus avoiding fetal asphyxia.
3. When the membranes rupture the fluid washes the birth canal from above downwards thus removing away any infectious material.

Note: The fetus swallows & inspires amniotic fluid and urinates in the amniotic sac.

AMNIOCENTESIS

I) DIAGNOSTIC AMNIOCENTESIS

Examination of amniotic fluid can be used for diagnosis of:

1. Severity of erythroblastosis fetalis by estimation of bilirubin content of amniotic fluid.
2. Chromosomal anomalies as *Down syndrome* (mongolism or trisomy 21).
3. Metabolic disorders as cystinuria (by examination of fetal cells for absent enzymes).
4. Genetic disorders as alpha and beta thalassemia (by analysis of DNA which makes up the chromosomes).
5. Open neural tube defects as anencephaly and open spina bifida (high level of alpha-fetoprotein).
6. Determination of fetal sex to prevent sex-linked diseases, for example a male fetus can be aborted when the mother is a carrier of haemophilia.
7. Assessment of fetal lung maturity. If lecithin:sphingomyelin ratio is 2 or more respiratory distress syndrome is not liable to occur if pregnancy is terminated. Also if phosphatidylglycerol (PG) is detected in amniotic fluid the fetal lung is considered mature.

II) THERAPEUTIC AMNIOCENTESIS

1. Induction of abortion in second trimester using 20% saline or 40% urea solution or prostaglandins.
2. To terminate pregnancy in intrauterine death using same technique.
3. Intrauterine transfusion in erythroblastosis fetalis.

TECHNIQUE

Amniocentesis is performed between 14–16 weeks of pregnancy. However, it can be done as early as 12 weeks. With early amniocentesis the number of fetal cells may be insufficient to allow successful cell culture. It needs 7 to 10 days to culture cells for genetic studies. Local anaesthesia is not necessary. A special needle 3–6 inches long is inserted into the amniotic sac through the abdominal wall guided by ultrasound and 1–1.5 ml of liquor for each week of pregnancy is withdrawn.

COMPLICATIONS

- a) *Fetal*: Injury to the fetus, umbilical cord and placenta. Trauma to the placenta can cause maternal isoimmunization and haemolytic disease in the fetus. After amniocentesis the mother is given anti-D serum (300 micrograms) if she is Rh-negative and not sensitized.
- b) *Maternal*: (1) Abortion (about 0.5%); (2) rupture of membranes; (3) preterm labour; (4) chorioamnionitis; (5) trauma leading to haemorrhage or bowel injury.

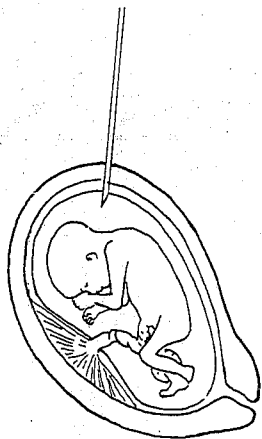


Fig. 9. Amniocentesis

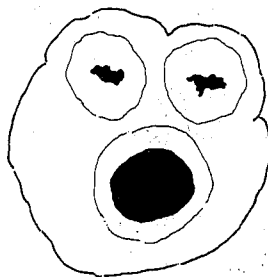


Fig. 10. Umbilical arteries and vein

THE UMBILICAL CORD (FUNIS)

STRUCTURE

At term the cord is 50–70 cm in length and 2 cm in diameter. It consists of mucoid connective tissue known as Wharton jelly covered by amnion. It contains one umbilical vein, two umbilical arteries, remnant of the yolk sac and remnant of the allantois. The umbilical vein carries oxygenated blood from the placenta to the fetus, while the umbilical arteries carry reduced blood from the fetus to the placenta.

ABNORMALITIES OF THE UMBILICAL CORD

1. *Abnormal attachment* (see before).
2. *Short cord*: Cord 32 cm or less. Shortening of the cord may be absolute or relative due to looping of the cord around the fetus. This may cause the following complications: (a) malpresentation, as transverse lie; (b) delay in descent of the fetus during labour; (c) premature separation of the placenta and accidental haemorrhage, (d) rupture of the cord and fetal asphyxia; (e) inversion of uterus; (f) failure of external cephalic version.
3. *Long cord*: Length is 100 cm or more. This may lead to: (a) coiling of the cord around the fetus; (b) true knots; (c) cord presentation and cord prolapse.
4. *Knots of the cord*: (a) *true knot*: formed when the fetus passes through a loop of the cord. When tight it causes fetal asphyxia and even death, (b) *false knot*: due to localized collection of Wharton jelly. It does not interfere with fetal circulation.
5. *Torsion of the cord*. If twisting is severe, fetal asphyxia may result.
6. *Congenital umbilical hernia*.
7. *Single umbilical artery*: It leads to congenital fetal malformation in 25–50% of cases.
8. *Other abnormalities* as tumours (myxoma and sarcoma), cysts (cyst from the allantois).

FETAL CIRCULATION

Oxygenated blood from the placenta is returned to the fetus by the umbilical vein, which passes through the umbilicus and runs in the free border of the falciform ligament, to reach the liver. The vein gives several branches to the hepatic sinuses. The main trunk, however, joins the left branch of the portal vein which is connected with the inferior vena cava by the ductus venosus. The inferior vena cava now contains a mixed stream consisting of oxygenated blood from the placenta, and venous blood from the lower half of the body. The inferior vena cava opens into the right atrium. The large portion of the blood (mainly oxygenated) passes through the foramen ovale to the left atrium. The smaller stream mixes with the venous blood coming from the head and neck through the superior vena cava and passes with it into the right ventricle. From the right ventricle the blood passes into the pulmonary trunk; most of it passes through the ductus arteriosus to the aorta while only a small portion passes through the pulmonary circulation to be returned by the pulmonary veins to the left atrium. The ductus arteriosus joins the left pulmonary artery and the terminal part of the aortic arch distal to the origin of the big vessels.

From the left atrium the blood passes through the mitral valve into the left ventricle, whence it is forced into the aorta. From the aorta the blood is distributed to the various organs and tissues of the fetus, but finally it reaches the umbilical artery which is a branch of the internal iliac artery. The umbilical arteries pass through the umbilicus into the cord to the placenta.

Immediately after delivery, certain changes occur in the fetal circulation. After cessation of the placental circulation, the umbilical vessels become functionless, the umbilical vein becomes obliterated forming the ligamentum teres, the umbilical arteries are obliterated to form the hypogastric ligaments and the ductus venosus forms the ligamentum venosum. The onset of fetal respiration after birth leads to establishment of the pulmonary circulation. The pressure in the left atrium is increased owing to the increased venous return from the lungs, while the pressure in the right atrium falls due to cessation of the placental circulation. The pressure on either side of the foramen ovale is

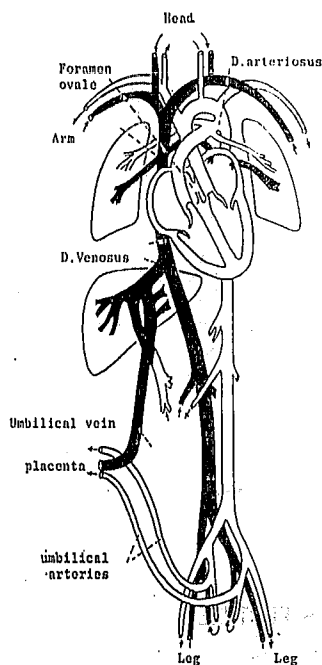


Fig. 11. Fetal circulation.

thereby equalized and the flap valve is closed. With the diversion of most of the blood into the lungs the ductus arteriosus becomes obliterated to form the ligamentum arteriosum.

Note: Embryo is the name given to the conceptus during the first 10 weeks of pregnancy. After this the word fetus is used.

MATERNAL CHANGES DURING PREGNANCY

"MATERNAL ADAPTATION TO PREGNANCY"

I) THE GENITAL ORGANS

A) Vulva

It becomes soft and violet in colour due to increased vascularity. There is liability to develop oedema and varicose veins.

B) Vagina

It becomes soft, warm and violet. Violet coloration of the vagina is called Chadwick or Jacquemier sign. Liability to develop varicose veins.

C) Cervix

It becomes enlarged, violet and soft (Goodell sign). Softening may be absent in the multigravida due to fibrosis. The cervical secretion is increased and part of it collects in the cervical canal to form a plug of mucus closing it mechanically (cervical operculum). Hormonal erosion (cervical ectopy) is common (due to hypertrophy and eversion of endocervical epithelium).

D) The Uterus

1. **Weight:** It is increased from 50 gm before pregnancy to become 1 kg at full term.
2. **Shape:** The nonpregnant uterus is pear shaped. At 8th week it is globular and the size of an orange, at 12th week, it is still globular and the size of a grape fruit or fetal head at term. At 16th week it becomes pyriform again and remains as such until the end of pregnancy.
3. **Size:** At 12 weeks the fundus is at the upper border of symphysis pubis, at 16 weeks it is at the junction of the lower third and upper two thirds of a line joining the umbilicus and upper border of symphysis pubis. At 20 weeks it is at the junction of the lower 2/3 and upper 1/3 of the above line. At 24 weeks, at the umbilicus, at 28 weeks; junction of lower 1/3 and upper 2/3 of a line joining the umbilicus and xiphoid cartilage. At 32 weeks; junction of lower 2/3 and upper 1/3 of the above line. At 36 weeks; it reaches

the xiphoid cartilage. At 40 weeks the fundus descends to a lower level (= 32 w) due to engagement of the head.

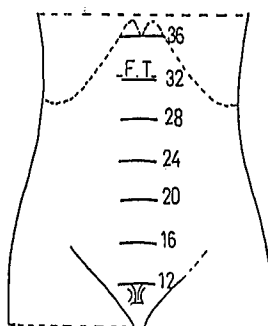


Fig. 12. Fundal level in weeks during pregnancy

F.T. = Full term

4. *Position:* During pregnancy the uterus is usually dextroflexed (deviated to the right) and dextrorotated (twisted on itself from left to right) and so the left round ligament becomes nearer to the midline (80%). Dextrorotation is caused by the presence of sigmoid colon on the left side of the pelvis.
5. *Consistency:* The uterus shows progressive softening due to increased vascularity and presence of amniotic fluid.
6. *Changes in the myometrium:*
 - a) Hypertrophy (increase in the size of the muscle fibre which is oestrogen effect).
 - b) Hyperplasia (increase in the number of muscle fibres which is progesterone effect).
 - c) Mechanical stretching.
 - d) Uterine contractions: During pregnancy the uterus undergoes intermittent contractions. When the uterus contracts it becomes hard and well defined, during relaxation it is soft, ill-defined and may be impalpable for a time. When these contractions are detected on bimanual examination, early in pregnancy while the uterus is still a pelvic organ we call them Palmer sign. Late in pregnancy, these contractions can be detected by abdominal examination and are called Braxton-Hicks contractions. The contractions help placental circulation.
7. *Formation of the lower uterine segment:* During pregnancy the uterus becomes differentiated into an upper and a lower uterine segment. The lower segment is developed from the uterine isthmus and lower part of uterine body which is covered by loose peritoneum. It is formed after the 12th week. During pregnancy, the lower segment undergoes gradual stretching and at term it is about 10 cm or 4 inches in length. It is

more developed during labour, and in case of obstructed labour it becomes markedly stretched and thinned and may even rupture.

<i>Upper Uterine Segment</i>	<i>Lower Uterine Segment</i>
1. Covered by adherent peritoneum.	1. Covered by loose peritoneum.
2. Muscle layer is thick. The fibres are arranged in 3 layers, outer longitudinal, inner circular and middle interlacing fibres forming figures of 8 around the blood vessels to control bleeding (living ligatures).	2. Muscle layer is thin. Fibres are arranged in 2 layers, outer longitudinal and inner circular.
3. The decidua is well developed.	3. Decidua is poorly developed.
4. Fetal membranes are firmly attached.	4. Membranes are loosely attached and separate during labour to form the bag of forewaters.
5. It is active so it contracts and retracts during labour.	5. It is passive so it dilates and stretches during labour.

8. *Retraction ring*: It is the groove found at the junction of the thick upper and thin lower uterine segment. During normal labour it does not exceed the upper border of the symphysis pubis and is called the physiological retraction ring. When labour becomes obstructed the retraction ring rises to a high level reaching the umbilicus or even higher and is called the pathological retraction ring or ring of Bandl.
9. *Blood supply*: The uterine blood flow increases progressively and reaches 500–700 ml/minute at term.

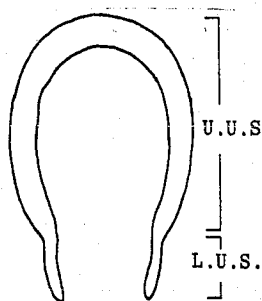


Fig. 13. Physiological retraction ring

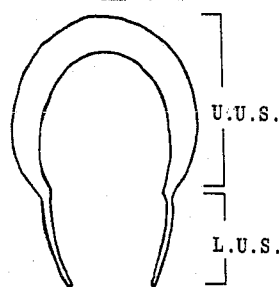


Fig. 14. Pathological retraction ring

E) The Fallopian Tubes

- Become elongated, stretched and vascularity is increased.

F) The Ovaries

Become more vascular, oedematous and enlarged, especially the one containing the corpus luteum of pregnancy. This degenerates at the 12th week and the placenta starts to perform its function which is production of oestrogens and progesterone. Relaxin is a hormone secreted by the corpus luteum of the pregnant woman, the chorion (placenta) and uterine decidua. Secretion continues throughout pregnancy.

II) THE BREASTS

- *First month:* Increase in size and sensitivity of the nipple and breast. Increased vascularity and subcutaneous dilated veins may appear.
- *Second month:* Increased pigmentation of the nipple and primary areola and increased prominence of Montgomery tubercles. These are enlarged sebaceous glands and are 12–20 in number. They appear as non pigmented nodules in the primary areola.
- *Third month:* Secretion of colostrum.
- *Fifth month:* The secondary areola appears. It is a reticulum of pigmentation appearing around the primary areola. About the same time striae gravidarum may appear. The breast changes are of diagnostic importance in the primigravida. In multigravida the signs may result from a previous pregnancy. Breast changes are not a sure sign of pregnancy because they may occur with fibroids or oestrogenic ovarian tumours.

N.B.: Colostrum is a thick yellowish secretion and differs from milk in that it contains more protein, less fat and less sugar. It contains colostrum corpuscles which are shedded cells from the gland acini containing fat droplets.



Fig. 15. Primary areola



Fig. 16. Montgomery tubercles

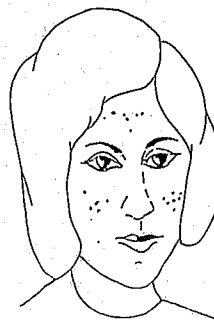


Fig. 17. Chloasma gravidarum

III) CUTANEOUS CHANGES

1. *Pigmentation of the skin*: This may occur anywhere, but the commonest sites affected are the nipple, areola, axilla and vulva. A dark brown line appears on the abdominal wall between the umbilicus and symphysis pubis and is known as linea nigra.
A butterfly pigmentation may appear on the cheeks and nose which is known as chloasma gravidarum or mask of pregnancy. These pigmentations may persist but the chloasma or melasma gravidarum usually disappears after delivery. Pigmentation is due to increased secretion of pituitary melanocyte-stimulating hormone, also oestrogen has a melanocyte-stimulating effect.
2. *Striae gravidarum*: Seen in about 50% of pregnant women. Usually appear in the second half of pregnancy. The striae appear as pink lines in the skin around the umbilicus and may also appear in the skin of the breast, thigh and buttock (areas of maximum stretch). They are caused by increased glucocorticoids or mechanical stretching causing separation of the collagen fibres in the dermis with exposure of the vascular subcutaneous tissue with appearance of these pink lines. After delivery they become white in colour and called striae albicans. The condition may occur in conditions other than pregnancy as Cushing syndrome (increased glucocorticoids), obesity from any cause and large ovarian cysts (mechanical stretching).
3. Signs of malnutrition as falling of hair may occur.
4. The blood flow through the skin and mucous membranes increases and leads to sensation of heat, increased sweating and nasal congestion which may cause epistaxis.

IV) CARDIOVASCULAR SYSTEM

1. *The heart*: In late pregnancy the apex beat is displaced upwards and outwards so that it lies in the 4th intercostal space outside the midclavicular line. This is due to elevation of the diaphragm by the pregnant uterus. The resting pulse rate increases 10–15 beats/minute. Functional systolic murmurs are common in normal pregnancy.
2. *The veins*: Liability to develop varicose veins due to:
 - a) Pressure of the gravid uterus on pelvic veins,
 - b) Relaxation of the smooth muscle fibres in the wall of the vessels by progesterone,
 - c) Increase in the blood volume,
 - d) Personal predisposition (congenital weakness).
3. *Blood pressure*: It remains normal in normal pregnancy. However, the pressure, both systolic and diastolic, may drop in the second trimester, but it becomes normal again in the last trimester. The systolic pressure falls 5 to 10 mmHg, and diastolic pressure 10 to 15 mmHg. This fall is due to vasodilatation which is caused by prostacyclin and hormones of pregnancy oestrogen and progesterone. Also the placenta acts as an

arterio-venous shunt and decreases the peripheral resistance. Prostacyclin is a prostaglandin secreted by the placenta, decidua, and endothelium of blood vessels. Levels of 140 mmHg systolic blood pressure or above, or 90 mmHg diastolic or above is abnormal.

4. *Blood volume*: It is increased during pregnancy reaching its maximum at 32 week (about 50% increase) and remains at this high level until delivery. The increase is more in the plasma volume than in the haemoglobin mass, leading to haemodilution and physiological anaemia. Anaemia is not physiological if the haemoglobin is less than 10 g/100 ml (70%).
5. *The cardiac output*: It is increased reaching its maximum at about 20 weeks when it is 30%-50% above the normal and the high level is maintained until delivery. It also increases during labour. It returns to nonpregnant level 2 weeks after delivery.
6. *Other changes*: Increase in the amount of fibrinogen (50%), in factors 7, 8, 9 and 10 and sedimentation rate. The number of erythrocytes and leucocytes increases (leucocytosis up to 12,000/mm³ is normal). Platelet count remains normal. Serum creatinine, blood urea nitrogen and uric acid decrease due to plasma volume expansion, and increased glomerular filtration rate leading to increased clearance of these substances. Serum lipids (cholesterol, triglycerides) increase. Calcium is slightly reduced due to haemodilution.

N.B.: The increase in fibrinogen contributes to the rise in sedimentation rate. Increase up to 50 mm in first-hour can be considered physiological during pregnancy.

V) RESPIRATORY SYSTEM

There is dyspnoea in late pregnancy due to elevation of the diaphragm. When the head becomes engaged in the pelvis in the last month, the dyspnoea is relieved.

VI) GASTROINTESTINAL TRACT

1. *Gingivitis*. The gums may become hyperaemic and soft, and may bleed when mildly traumatized, as with a tooth brush. A focal vascular swelling of the gums called epulis of pregnancy may develop but regresses spontaneously after delivery.
2. *Increased salivation* which may be troublesome (Ptyalism).
3. *Morning sickness*: It means nausea and sometimes vomiting especially in the morning. It occurs in about 50% of pregnant women. It usually appears at the 6th week and disappears after the 12th week.
4. *Changes of appetite* "longing or pregnancy picka". The patient desires certain foods, odors and other materials and dislikes others.

5. *Hypochlorhydria* is frequent and interferes with digestion of food and absorption of iron. It is due to regurgitation of alkaline chyle from intestine into the stomach.
6. *Indigestion and flatulence* due to hypochlorhydria and diminished gastric motility. Motility is diminished due to presence of progesterone or deficiency of the hormone motilin which stimulates peristalsis.
7. *Heartburn*, due to regurgitation of acid, sometimes bile from the stomach in the lower oesophagus. It is caused partly by pressure of the uterus on the stomach and partly by progesterone which relaxes the lower oesophageal sphincter (cardiac sphincter).
8. *Constipation* is common due to pressure of the uterus on the pelvic colon, relaxation of the smooth muscle fibres by progesterone and sedentary life during pregnancy. In some women iron supplementation may be the cause.
9. *Piles* may occur (due to the factors which lead to varicose veins plus constipation).
10. Tendency to *gallstone formation* due to atony and delayed emptying of the gallbladder.

VII) URINARY TRACT

1. *Bladder*: Frequency of micturition occurs in the first 3 months due to pressure on the bladder by the pregnant uterus, congestion of bladder mucosa also progesterone increases mucosal sensitivity. It usually disappears after the 12th week when the uterus becomes an abdominal organ. Frequency recurs in the last month when the head becomes engaged.
2. *Ureters*: Become elongated, dilated, and tortuous. This is due to the effect of progesterone, pressure of the uterus on the ureters against the pelvic brim, and there is also hypertrophy of the wall of the pelvic portion of the ureter leading to narrowing of the lumen. These changes are more marked on the right side due to right obliquity of the uterus and pressure of the dilated right ovarian vein while the left ureter is protected from pressure by sigmoid colon, and more common in the primigravida because the abdominal muscles are tense. Dilatation resolves within 6 weeks after delivery.
3. *Kidneys*: During pregnancy the kidney increases in length by 1–1.5 cm and so weight is increased. Liability to pyelonephritis of pregnancy due to stasis of urine which is caused by the changes in the ureters. The renal blood flow and glomerular filtration rate increase by about 50%. This leads to increased clearance of creatinine, blood urea nitrogen, and uric acid. It may also lead to glycosuria in about 15% of cases as the renal tubules can not reabsorb the extra amount of filtered glucose (physiological glycosuria).

VIII) NERVOUS SYSTEM

Functional changes may occur, especially in neurotic women: (1) morning sickness which may be neurotic in origin; (2) changes of appetite or longing; (3) during pregnancy, some women are sleepy and depressed, others may be irritable and suffer insomnia.

IX) MUSCULOSKELETAL SYSTEM

Progressive lordosis as the uterus enlarges. Increased mobility of pelvic joints due to softening and relaxation of ligaments by progesterone. Leg cramps may occur in the second half of pregnancy. They often appear at night (see minor disorders of pregnancy).

X) ENDOCRINE GLANDS

Pituitary

There is increase in the size of the anterior lobe (2-3 folds) due to increase in prolactin-secreting cells. Serum prolactin concentration increases. Serum FSH and LH are reduced due to high oestrogen and progesterone level. Posterior lobe does not hypertrophy.

Thyroid

Normal pregnancy may lead to mild thyroid enlargement which is caused by increased vascularity and hyperplasia of glandular tissue. The basal metabolic rate is increased by 20% above the normal in the last trimester and is due to increased oxygen consumption by the fetus and uterus. The changes in the gland are due to increased secretion of thyroid-stimulating hormone (TSH) and chorionic thyrotrophin (secreted by the placenta). Also HCG has a thyroid-stimulating effect. It stimulates the TSH receptors on the thyroid gland.

Parathyroid Glands

Undergo hypertrophy due to increased demand for calcium during pregnancy. The parathormone level is increased to increase calcium absorption by the mother.

Adrenals

There is increased activity evidenced by skin pigmentation and impaired carbohydrate metabolism. There is increase in aldosterone and cortisol (both combined and free).

Ovaries: See page (14).

Placenta

The hormones secreted by the placenta have been discussed on page (3).

XI) METABOLIC CHANGES

1. *Increase in body weight* by 9–12 kg throughout pregnancy. The increase in weight during pregnancy is attributable to the weight of uterus and its contents, enlarged breasts, increased blood volume and interstitial fluid and increased body fat and protein (maternal reserve).
2. *Retention of water, sodium, potassium and chloride*. This explains the diuresis which occurs in the first 2 days after delivery.
3. *Protein metabolism*: There is nitrogen retention for formation of the fetus and maternal tissues. This will reduce the blood urea.
4. *Carbohydrate metabolism*: It is disturbed during pregnancy. Pregnancy has a diabetogenic effect. Diabetes may appear only during pregnancy or become aggravated by pregnancy because human placental lactogen, oestrogens, progesterone, prolactin and cortisol antagonise insulin. Also the placental enzyme insulinase destroys insulin and may play a role. Physiological glycosuria may occur (see diabetes mellitus). The fasting blood glucose is lowered due to transfer of glucose to the fetus.
5. *Fat metabolism*: Serum lipids as cholesterol and triglycerides are increased. The cause of hyperlipidaemia is unknown.
6. There is *increased demand for calcium, iron and all vitamins*. Iron is stored in the fetal liver during the last three months of pregnancy because the mother's milk is poor in iron. This store is sufficient for the first 6 months after delivery.

MINOR DISORDERS OF PREGNANCY

During pregnancy a number of minor complaints may arise. These are annoying but do not disable the pregnant woman. The majority of these symptoms are the result of large amounts of placental hormones particularly oestrogen and progesterone.

I) GASTROINTESTINAL DISORDERS

1. *Gingivitis*. The gums may become hyperaemic and soft, and may bleed when mildly traumatized, as with a tooth brush. Epulis of pregnancy may develop. Treated by dental hygiene and cryosurgery for severe cases.
2. *Ptyalism*, which is increased salivation may be troublesome. It is due to failure to swallow saliva and not due to increase in amount. Smoking is stopped and anticholinergic drugs may help.
3. *Morning sickness*. See vomiting with pregnancy.
4. *Indigestion and flatulence* due to hypochlorhydria (caused by regurgitation of alkaline chyle from intestine into the stomach) and diminished gastric motility (caused by progesterone or deficiency of motilin).
5. *Heartburn* (oesophageal reflux), due to regurgitation of acid, sometimes bile from the stomach in the oesophagus. It is caused partly by pressure of the uterus on the stomach and partly by progesterone which relaxes the lower oesophageal sphincter. The treatment includes (a) small frequent meals to prevent overdistension of the stomach. The evening meal should be taken at least 3 hours before going to bed, (b) avoid fatty foods, chocolate, and smoking, as these relax the lower oesophageal sphincter, (c) the bed should be raised at the head (15–20 cm), and an extra pillow is used, (d) antacids. Preparations containing aluminium hydroxide are favoured as they are not absorbed and alkalosis is unlikely. Calcium containing antacids are contraindicated as calcium relaxes the sphincter.
6. Atony and delayed emptying of the gallbladder with tendency to stone formation.
7. *Constipation*. For aetiology see maternal changes during pregnancy. It is treated by (a) evacuation of the bowel at the same time each day, and do not neglect call to stool (bowel training), (b) diet rich in fibre in the form of vegetables, fruits, and bran and reduce sugar, (c) milk and avoid dehydration by increasing fluid intake, (d) minimize coffee and tea as they are diuretics and cause dehydration, (e) increase physical activity and avoid sedentary life, (f) a mild laxative may be needed. Liquid paraffin is better avoided as it prevents absorption of fat soluble vitamins. In some women iron supplementation may be the cause.
8. *Piles*. For the aetiology see maternal changes during pregnancy. Treat constipation plus local ointments and suppositories.

II) VARICOSE VEINS

For the aetiology see maternal changes during pregnancy. About 10–20% of pregnancy women suffer varicose veins of the legs. Varicosities of the vulva and vagina are less common (about 3% and 2% respectively) and tend to occur in parous women in late pregnancy. Varicose veins lead to discomfort, oedema, and non-cosmetic appearance. Complications include thrombophlebitis usually in the puerperium, haemorrhage, and ulceration. The treatment is (a) avoid long periods of standing and encourage active exercise, (b) avoid constricting clothes, (c) keep the legs elevated while sitting and during sleep, (d) use of elastic stockings. These should be removed at night and applied with leg elevated before getting out of bed in the morning (empty veins), (e) stretch panties may be necessary for vulval varicosities.

III) URINARY SYMPTOMS

Frequency of micturition occurs in the first 3 months and recurs in the last month when the head becomes engaged. Stress incontinence may occur.

IV) MUSCULOSKELETAL DISORDERS

1. *Backache*. The majority of pregnant women complain of low backache which increases as pregnancy advances. It is due to increased lumbar lordosis to counter-balance the forward growth of the uterus (pride of pregnancy). This puts strain on ligaments and muscles leading to pain. Strain of sacroiliac joint is relatively common. Progesterone causes softening and relaxation of ligaments. Backache is treated by: (a) more periods of rest, (b) use of maternity corset, (c) local heat in the form of hot water bag or infrared lamp, (d) analgesics given systemically or as local creams. Paracetamol is the drug of choice. Non-steroidal anti-inflammatory drugs as indomethacin may be given, but contraindicated after 34 (or 32) weeks of pregnancy to avoid premature closure of the ductus arteriosus, (e) physiotherapy may be needed. Orthopaedic consultation is indicated if pain is severe, or radiates to the legs, and in the presence of neurological signs. Following pregnancy the condition may ameliorate slowly.
2. *Leg cramps*. These are common in the second half of pregnancy particularly at night. The exact cause is unknown. It may be related to shift of blood away from the muscle, i.e., ischaemic cramp, or it may be tetanic cramp caused by lack of calcium, or increased phosphorous, or both. Treated by taking calcium tablets, and reducing the intake of phosphorous-containing substances as milk, meat, and cheese. Vitamin B complex may be tried. Leg massage and hyperextension of foot help during the attack.
3. *Round ligament strain*. Pain is felt along the round ligament and in the groin. Pain is usually unilateral and left-sided, due to dextroflexion of the uterus. It is due to

stretching of the nerve fibres in the round ligaments. The thickened ligaments may be felt lateral to the uterus in the lower abdomen.

V) NERVOUS SYSTEM DISORDERS

1. *Headache*. It is relatively common, and attributed to intracranial vasodilatation caused by oestrogen and progesterone. It also occurs in women taking contraceptive pills. It is most troublesome in the second trimester, but may persist throughout pregnancy. However, headache may be due to lack of sleep, or overwork. An analgesic is prescribed.
 2. *Fainting*. It results from lowering of blood pressure due to vasodilatation which occurs in pregnancy.
 3. *Insomnia*. During pregnancy some women are sleepy and depressed, others may be irritable and suffer insomnia.
 4. *Carpal tunnel syndrome*. Caused by compression of the median nerve as it passes through its fibrous tunnel at the wrist, as a result of fluid retention and oedema of pregnancy. There is tingling, numbness and burning sensation affecting the radial half of the hand. Treatment includes reassurance, use of a wrist splint, diuretics, non-steroidal anti-inflammatory drugs, and local injection of hydrocortisone in the tunnel below the fibrous roof (retinaculum). Operation is rarely needed during pregnancy by incising the retinaculum to relieve compression.
- Other compression neuropathies affect the lateral cutaneous nerve of the thigh, obturator and peroneal nerves.

VI) CUTANEOUS CHANGES

1. *Striae gravidarum*. See maternal changes during pregnancy.
2. The blood flow increases through the skin, and mucous membranes, and leads to sensation of heat, excessive sweating, and nasal congestion which may cause epistaxis.

VII) LEUCORRHOEA

The normal vaginal discharge increases during pregnancy because of excessive oestrogen and may form a complaint. However, a pathological discharge, e.g., monilial infections which is common in pregnancy must be excluded.

DIAGNOSIS OF PREGNANCY

The diagnosis depends upon symptoms, signs and investigations.

DIAGNOSIS IN THE FIRST TRIMESTER (FIRST 12 WEEKS)

I) Symptoms

1. Amenorrhoea: It is due to the presence of a high level of oestrogen and progesterone without withdrawal. It is not a sure evidence of pregnancy because it may be caused by other conditions as lactation. Also bleeding may occur in the early months of pregnancy and may be mistaken for menstruation, e.g. abortion.
2. Morning sickness.
3. Frequency of micturition.
4. Breast symptoms as enlargement, heaviness and pain.
5. Changes of appetite or longing.
6. Some women are sleepy and depressed, others may be irritable and suffer insomnia.

II) Signs

1. *Breast signs*: see page (14).
2. *Uterine signs*:
 - a) The uterus is enlarged and soft in consistency. At 8th week it is the size of an orange. At 12th week it is the size of a grape fruit or fetal head at term with the fundus at the upper border of symphysis pubis.
 - b) Palmer sign: Uterine contractions followed by relaxation felt during bimanual examination.
 - c) Hegar sign: Two fingers are placed in the anterior vaginal fornix in front of the cervix and the other hand is placed abdominally behind the uterus. If the internal and external fingers can be approximated, the sign is positive and means that the lower uterine segment is soft and empty. It is positive between 6 and 12 weeks of pregnancy. Before the 6th week the uterus is not soft enough. After the 12th week the fetus fills the whole uterine cavity. It is not a sure sign of pregnancy because it can be obtained in other conditions with a soft uterus as metropathia haemorrhagica, also it is present in only two-thirds of pregnant cases.

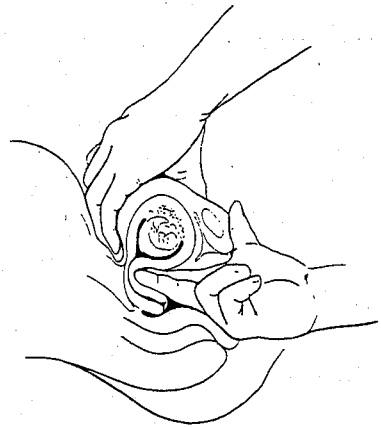


Fig. 18. Hegar sign

3. *Cervical signs*: The cervix is enlarged, violet and soft.
4. *Vaginal signs*: The vagina is soft, warm and violet.
5. *Vulval signs*: The vulva is soft and violet.

III) SPECIAL INVESTIGATIONS

(1) Pregnancy tests; (2) Sonography

Pregnancy Tests

These depend on the presence of human chorionic gonadotrophin (HCG) in blood or urine of the pregnant woman.

Urine Tests

These include agglutination and enzyme immunoassay tests.

- a. *Agglutination tests*. Depend on the use of latex particles or sheep red cells coated with HCG and animal serum (horse or rabbit) containing antibodies against HCG. Agglutination tests detect HCG if its concentration in urine is 500–800 mIU/ml. The test is reliable in 98% of cases and becomes positive about one week after the first missed period, i.e. the 5th week of pregnancy. In the slide test (as Gravindex test) a drop of morning urine is mixed on a slide with a drop of animal serum containing antibodies against HCG. Mix for one minute, then add one drop of latex particles coated with HCG and mix again for one minute. Failure of agglutination of latex particles means a positive test and the patient is pregnant because the added animal serum was neutralized by HCG present in urine. If agglutination occurs the woman is not pregnant. The test is simple and the result is obtained within 2 minutes. When sheep red cells are used (e.g. Pregnosticon test) the test is carried out using a test tube and the result is obtained within 2 hours but is more sensitive.
- b. *Enzyme immunoassay tests*. The enzyme-linked immunoabsorbent assay tests (ELISA tests) are sensitive and detect small amounts of HCG (10–50 mIU/ml) so pregnancy is diagnosed before the first missed period by 5–7 days. Some tests give the result in 5 minutes. The ELISA tests detect the whole molecule of HCG in urine or serum.

Blood Test

Detection of beta-subunit of HCG in serum by radioimmunoassay is a very sensitive test and detects small amounts of the hormone (one mIU/ml of beta-subunit). It can diagnose pregnancy before the first missed period by 5–7 days (becomes positive one day after implantation).

Note: mIU/ml = milli international unit per millilitre.

Sonography

It means the use of sound waves above the range of human hearing hence the term ultrasonic technique. By transvaginal sonography, the pregnancy sac appears as a white ring at 4 weeks of pregnancy, the fetal pole at 5 weeks, and fetal heart movement at 6 weeks. Transabdominal ultrasound is less sensitive with about one week difference. Also the fetal heart sounds can be heard after the 10th week by the Doppler stethoscope (Doptone or Sonicaid).

N.B.: Pregnancy tests are used for diagnosis of pregnancy, ectopic pregnancy, missed abortion, intrauterine death, hydatidiform mole and choriocarcinoma.

The causes of a false-positive urinary pregnancy test

(1) Proteinuria (prevents agglutination of HCG with anti-HCG antibodies); (2) haematuria as haemoglobin is a protein and may give a false positive reaction; (3) tumours producing human chorionic gonadotrophin as choriocarcinoma; (4) lesions of hypothalamus and pituitary gland and drugs as penicillin and phenothiazines which stimulate release of luteinizing hormone from anterior pituitary; (5) immunologic diseases as systemic lupus erythematosus because immunoglobulin M interacts with test reagents; (6) premature menopause (due to cross-reaction with elevated luteinizing hormone); (7) urine which is excessively alkaline; (8) at time of ovulation due to elevated LH; (9) HCG injection used for treatment of infertility.

The causes of a false-negative urinary pregnancy test

(1) Test done in early pregnancy; (2) diluted urine due to excessive fluid intake and diuretics; (3) urine stored too long at room temperature.

DIAGNOSIS IN THE SECOND TRIMESTER (13-28 WEEKS)

I) Symptoms

1. Amenorrhoea.
2. Breast symptoms which become more marked.
3. Quickening: It is the first time at which the mother feels fetal movements. In multi-gravidae, it usually occurs between the 16th and 18th week. In primigravidae, it usually occurs between the 18th and 20th week. This difference is due to previous experience. Intestinal movements may be mistaken for fetal movements.
4. Progressive abdominal enlargement.
Morning sickness and frequency of micturition normally disappear after the 12th week.

II) Signs

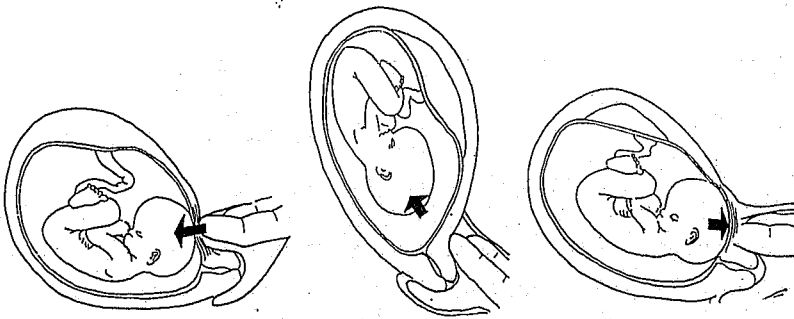
1. Breast signs.

2. Uterine signs:

- a) The uterus is felt abdominally as a soft or cystic swelling with a convex upper border. It undergoes intermittent contractions which can be felt by abdominal palpation (Braxton-Hicks contractions).
- b) Uterine souffle: It is a soft blowing murmur synchronous with the maternal pulse. It is due to flow of blood in the dilated uterine arteries (may be heard in presence of fibroids).

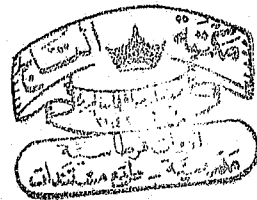
3. Fetal signs:

- a) Ballottement: This sign depends on the presence of a solid body, that is the fetus moving in a fluid medium (the amniotic fluid). There are two types:
 - i) *Internal ballottement*: This can be detected between the 16th and 28th week. (The patient lies in the dorsal position, press the fundus by one hand, while the 2 fingers of the other hand are placed into the anterior vaginal fornix, give a gentle upward push with these fingers. The head will move away and after a moment, it falls back giving a touch to the fingers). The sign cannot be detected before the 16th week because the fetus is too small, and not after the 28th week because it is large relative to the amount of the liquor.
 - ii) *External ballottement*: Detected after the 24th week (the 2 hands are placed abdominally on both sides of the uterus and give sharp pushes with the fingers of one hand).



- Fig. 19. Internal ballottement

- b) Palpation of fetal parts as early as the 24th week.
- c) Feeling or seeing fetal movements.
- d) Auscultation of fetal heart sounds (FHS).



- e) Umbilical or funic souffle: It is a sharp whistling sound synchronous with the fetal heart rate. It is due to flow of blood in the umbilical arteries. It is occasionally heard when a loop of the cord lies below the stethoscope behind the anterior uterine wall. It is a sure sign of pregnancy.
4. Appearance of striae gravidarum and linea nigra.

III) SPECIAL INVESTIGATIONS

Done in doubtful cases because diagnosis of pregnancy is usually easy in the second trimester.

1. Pregnancy tests.
2. Sonar or X-ray examination: The latter shows parts of fetal skeleton at 16th week and the whole skeleton at 24th week. Sonar is preferred to avoid hazards of irradiation. In fact X-ray examination is resorted to if ultrasound is not available.

DIAGNOSIS IN THE THIRD TRIMESTER (29-40 WEEKS)

The diagnosis is easy because all the sure signs of pregnancy are present and these are: (1) Palpation of fetal parts; (2) palpation of fetal movements. (3) hearing FHS; (4) hearing umbilical souffle; (5) seeing fetal movements; (6) seeing fetal skeleton by sonar or X-ray.

DIFFERENTIAL DIAGNOSIS OF EARLY PREGNANCY

1. Other causes of amenorrhoea.
2. Causes of symmetrical enlargement of the uterus as submucous fibroid.

DIFFERENTIAL DIAGNOSIS OF LATE PREGNANCY

1. Causes of pelvi-abdominal swellings as fibroids or ovarian cyst.
2. Pseudopregnancy or pseudocyesis (imaginary pregnancy): False pregnancy may occur in women near menopause or in women anxious to become pregnant. This leads to psychological disturbance affecting the hypothalamus. There is amenorrhoea, sometimes morning sickness and progressive abdominal enlargement which is due to deposition of fat in the abdominal wall and gaseous distension of the bowel. Intestinal movements may be mistaken for fetal movements. If the condition is not diagnosed the patient will suffer abdominal pain at the expected date of delivery which is similar to labour pain (spurious or false labour). The uterus is normal in size but it may not be felt because of abdominal distension. The pregnancy test is negative and ultrasound shows empty uterus.

ANTENATAL CARE

It is the medical and psychological supervision of the pregnant woman so that she will be able to go through pregnancy, labour and puerperium without complication to herself or her baby. The antenatal care consists of: (1) History taking; (2) physical examination; (3) special investigations; (4) instructions or advice to the pregnant mother; (5) reassurance.

HISTORY

1. Personal history

Name, age, duration of marriage, number of marriages if present, occupation, address, and telephone number.

2. Complaint

It is taken in the patient's own words. Sometimes the only complaint is amenorrhoea and the patient is coming for antenatal care.

3. Present history

Each complaint is taken in detail. The patient is also asked about other symptoms as headache, visual disturbances, gastrointestinal symptoms as nausea, vomiting, diarrhoea or constipation, urinary symptoms as burning pain and frequency, vaginal bleeding, vaginal discharge and itching as well as the presence of oedema.

4. Past history

- a) Past medical history. Ask about the presence of any chronic disease as hypertension, heart disease, diabetes mellitus, renal disease, syphilis, etc.
- b) Past surgical history. Ask about any operation done for the patient.
- c) Hormonal treatment as intake of contraceptive tablets.
- d) History of blood transfusion.
- e) The presence of allergy to drugs, food, etc.

5. Family history

Ask about conditions that can be inherited by the mother as hypertension, diabetes mellitus, pre-eclampsia in mother or sister, twins, and congenital fetal anomalies.

6. Menstrual history

Menarche, duration of menstrual bleeding, length of cycle (D/C), amount (slight, moderate or excessive), the presence of pain and its relation to bleeding, intermenstrual bleeding or discharge, the first day of last menstrual period (LMP) and the expected date of delivery (EDD).

7. Obstetric history

- a) In each delivery we ask about: (1) Duration of pregnancy (preterm, full term or postterm); (2) mode of delivery (spontaneous vaginal delivery, by forceps, etc.); (3) the newborn (male or female, alive or dead); (4) fetal or maternal complication in the puerperium; (5) time of last delivery.
- b) In each abortion we ask about: (1) Duration of pregnancy; (2) mode of termination (spontaneous or induced); (3) complication in the postabortive period; (4) time of last abortion.

8. Social history

Ask about special habits of medical importance as smoking, alcohol and drugs.

PHYSICAL EXAMINATION

It is general, abdominal, and vaginal.

General Examination

- General condition (good, moderate or bad).
- Height; Weight; Gait.
- Vital signs: Pulse; Temperature; Blood pressure.
- Eye examination: Pallor; Jaundice; Oedema of eye lids.
- Nose: e.g. saddle nose due to syphilis.
- Cheeks: for cloasma gravidarum.
- Mouth: Tongue; Teeth; Tonsils.
- Neck: Thyroid gland; Lymph glands; Congested neck veins.
- Chest: Heart; Lungs; Breasts.
- Lower limbs: Varicose veins; Bony deformities.

Abdominal Examination

A) Inspection

1. Size: If abnormally huge it suggests multiple pregnancy or polyhydramnios.
2. Shape: Pendulous abdomen in a primigravida suggests contracted pelvis.
3. Scars: As caesarean section scar.
4. Striae gravidarum.
5. Suprapubic hair: Feminine with an upper straight border, or masculine with a convex upper border which suggests android pelvis.
6. Pigmentation as linea nigra.
7. The presence of hernia.

B) Palpation

1. Fundal Level

The obstetrician stands on the right side of the patient and the ulnar border of the left hand is moved from the xiphoid cartilage downwards until the resistance of the fundus is felt. This gives idea about the duration of pregnancy in weeks and is compared with the period of amenorrhoea.

2. Fundal Grip

Still on the right side of the patient and looking to her face the fundus of the uterus is palpated with both hands to know the part of the fetus occupying it. The head is felt small, hard, regular, ballottable, separated from the trunk by a groove and it is tender (uterine wall is compressed by the hand against the hard head). The breech is large, soft, irregular, not ballottable, not separated from the trunk by a groove and it is not tender.

3. Umbilical or Lateral Grip

The hands are placed on both sides of the uterus at the level of the umbilicus to determine the position of the back and the limbs. Fix the fetus with one hand, and feel with the other hand. This is done alternately. The back is felt as a firm, convex, smooth surface. The limbs are felt as small, irregular, mobile, slippery knobs. The back is then followed downwards until the groove of the neck is reached, the projection just above this groove is the anterior shoulder at which we hear the fetal heart sounds. In case of transverse or oblique lie the head is felt on one side of the midline and the breech on the opposite side.

4. The First Pelvic or Pawlick Grip

The right hand is placed on the symphysis pubis with the thumb parallel to the right inguinal ligament and the four fingers parallel to the left ligament. Approximate the thumb and fingers together to feel the part of the fetus occupying the lower uterine segment. If the head is engaged it cannot be felt by the first pelvic grip.

5. The Second Pelvic Grip

Look towards the feet of the patient and apply the hands on both sides of the lower abdomen. They are pushed towards the pelvic brim and are approximated at the same time. The aim of this grip: (a) to feel the head if it is engaged (b) to feel the head of another fetus in case of twins, (c) to know the position of the head. If it is flexed the occiput is felt at a lower level than the sinciput, if extended the occiput is felt higher and if the head is deflexed the occiput and sinciput are felt at the same level.

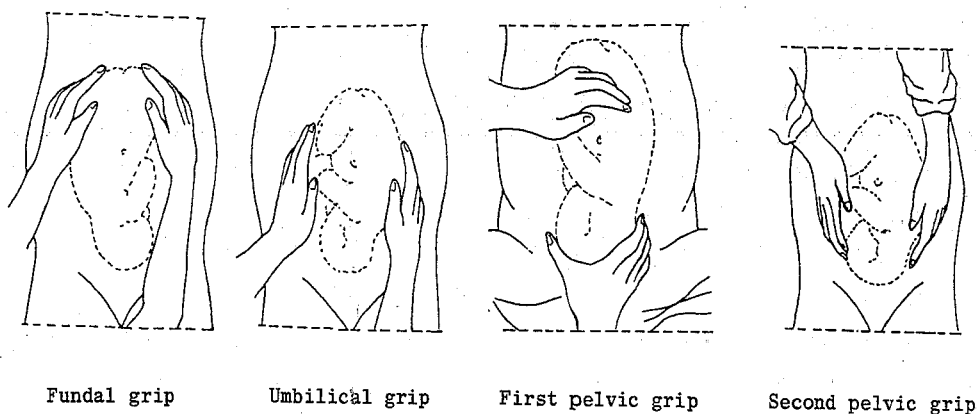


Fig. 20. Abdominal palpation

6. The rest of the abdomen is palpated to detect any abnormality.

Note: Leopold manoeuvres include fundal grip, umbilical grip, first and second pelvic grip.

C) Auscultation

The fetal heart sounds are heard by the fetal or Pinard stethoscope or by the Doppler stethoscope (Sonicaid or Doptone). They can be heard all over the abdomen with a point of maximum intensity over the anterior shoulder and therefore this point varies according to the position of the fetus. They are usually heard at the 24th week by the fetal stethoscope and at the 10th week by the Sonicaid. The fetal heart sound is a tic-tac rhythm with a rate between 120–160 beats per minute under normal conditions.

Value of Auscultating Fetal Heart Sounds

1. It is a sure sign of pregnancy.
2. To diagnose intrauterine fetal death.
3. To know position and presentation of the fetus.
4. Diagnosis of twin pregnancy.
5. Diagnosis of fetal distress.
6. To follow up the progress of labour.

Differential Diagnosis of Fetal Heart Sounds

- a) *Fetal Sounds*: (1) Umbilical souffle; (2) sounds resulting from fetal movements.
- b) *Maternal Sounds*: (1) Uterine souffle; (2) intestinal movements; (3) transmitted aortic pulsations.

N.B.: In occipito-anterior position the FHS are heard at the middle of a line joining the umbilicus and the midinguinal point. In occipito-posterior position they are heard near the anterior superior iliac spine, and in breech presentation they are heard above the umbilicus. In transverse lie they are heard on the side of the head.

VAGINAL EXAMINATION

It is performed during pregnancy in the following conditions:

1. In early pregnancy, at the first visit, to confirm the diagnosis and to exclude any pelvic lesion.
2. At the 36th week to do internal pelvimetry and cephalopelvic proportion.
3. When complication occurs as bleeding or discharge.

SPECIAL INVESTIGATIONS

1. Urine is tested for protein and sugar. Urine culture to diagnose asymptomatic bacteriuria.
2. Complete blood count, blood group and Rh. group.
3. Antibody screen test. Indirect Coombs test to detect antibodies against antigens on red blood cells in both Rhesus negative and positive women.
4. Screening for rubella antibodies.
5. Screening test for syphilis.
6. Hepatitis B surface antigen is tested for.
7. Screening test to diagnose gestational diabetes. Done between 24–28 weeks.
8. Papanicolaou cervical smear, if the last smear was taken more than one year ago.
9. Testing for human immune deficiency virus (HIV).
10. Other investigations according to need as pregnancy test.

DIAGNOSIS

The following points are necessary for complete diagnosis:

- | | |
|---|------------------------|
| 1. Gravidity | Gravida 1 |
| 2. Para X + Z | Para 0 + 0 |
| 3. Duration of pregnancy in weeks | More or less 36 weeks |
| 4. Lie | Longitudinal lie |
| 5. Presentation | Cephalic presentation |
| 6. Position | Left occipito-anterior |
| 7. Associated conditions as diabetes mellitus | Diabetes mellitus |

N.B.: X is the number of deliveries and Z is the number of abortions. Para 0 + 0 means no previous deliveries or abortions. Gravidity is the number of pregnancies. The lie, position and presentation are determined from the 32nd week onwards.

Causes of an oversized pregnant uterus (larger than the period of amenorrhoea)

1. Wrong calculation.
2. Bleeding in early pregnancy misdiagnosed as menstruation.
3. Large fetus.
4. Fetal malformations as hydrocephalus.
5. Multiple pregnancy.
6. Hydatidiform mole.
7. Polyhydramnios.
8. Tumours as fibroids or ovarian cyst.
9. Abruptio placentae with concealed haemorrhage.

Causes of an undersized pregnant uterus (smaller than the period of amenorrhoea)

1. Wrong calculation.
2. Pregnancy during a period of amenorrhoea e.g. lactation.
3. Small fetus.
4. Intrauterine fetal death.
5. Oligohydramnios.
6. Certain malpresentations as transverse lie or breech with extended legs.

INSTRUCTIONS TO THE PREGNANT MOTHER**A) Diet During Pregnancy**

1. *Kilocalories (kcal)*: About 2500 kcal are needed per day. In fact the pregnant woman needs 15% (300–500) more kcal per day. The most important is the quality and not the quantity of the diet.
2. *Proteins*: More proteins are needed and the amount is 2 g/kg body weight daily. Half of the amount must contain the essential amino acids (animal proteins). Protein deficiency may cause anaemia and oedema.
3. *Fats*: Should be restricted to avoid abnormal gain in weight.
4. *Carbohydrates*: The amount is not changed.
5. *Vitamins*: All the vitamins are needed in increased amounts during pregnancy:
 - Vitamin A: The pregnant mother needs 5000 I.U. daily.
 - Vitamin B group: All are essential; B6 deficiency may cause vomiting of pregnancy. Folic acid deficiency leads to megaloblastic anaemia in the mother, neural tube defects in the fetus and intrauterine growth restriction, daily requirement 0.5 mg.
 - Vitamin C: Deficiency leads to scurvy, and postpartum haemorrhage (100 mg/day).
 - Vitamin D: Deficiency leads to osteomalacia in the mother and rickets in the child (400 I.U. daily).

- Vitamin E: Deficiency may cause abortion in rat, however, this is not proven in human.
- Vitamin K: Deficiency leads to postpartum haemorrhage. It may cause haemorrhage in the fetus.

6. *Minerals:*

- a) **Iron:** Deficiency leads to anaemia of the mother. The fetus obtains his needs of iron even if the mother is iron deficient. Daily requirement 3–6 mg (1.5 mg for non pregnant which is absorbed from the normal diet). As this amount (3–6 mg) cannot be supplied by the normal diet, iron should be supplemented during pregnancy. If prophylactic iron is not given, up to 50% of pregnant women develop iron deficiency anaemia. Iron supplementation is started after the first trimester when nausea disappears.
 - b) **Calcium:** Deficiency leads to osteomalacia in the mother and rickets in the child. It predisposes to cramps, muscle twitches or pain in pelvic bones and back. The pregnant woman needs 1.2 g calcium/day. One litre of milk contains 1 g calcium.
 - c) **Iodine:** Deficiency may lead to congenital goitre and goitre of the mother.
7. *Smoking.* There are about 4000 compounds in tobacco smoke, among them are nicotine, carbon monoxide, thiocyanate, and many other harmful substances. Smoking increases the risk of abortion, ectopic pregnancy, premature rupture of membranes, preterm labour, intrauterine growth restriction, intrauterine death, placental abruption, placenta praevia, and diminished milk production during lactation (by about 30%). Smoking also increases the risk of sudden infant death syndrome (crib or cot death), and respiratory problems in the neonate and during childhood as bronchial asthma. It also affects physical growth and mental development of children up to the age of 10. In addition smoking leads to ptyalism, nervousness and hyperemesis gravidarum.
8. *Coffee and tea* are minimized during pregnancy. Caffeine present in coffee, tea, and chocolate reduces iron absorption and may cause anaemia in the mother. Coffee and tea are diuretics and lead to constipation as a result of dehydration. Other effects include anxiety and palpitations.

Note:

- **Folic acid supplementation.** Extra folic acid is recommended for all pregnant women at a dose of 400 micrograms daily before conception and up to 12 weeks of pregnancy. The dose is increased to 5 mg daily if there is a past history of neural tube defects, diabetes mellitus, and when the mother is receiving anticonvulsant therapy for epilepsy. This prophylactic supplementation reduces the incidence of neural tube defects by 50–70% but with a lesser percentage in obese women. Closure of the neural tube occurs between 3–4 weeks post-conception (5–6 weeks of pregnancy). So prophylaxis must be started before conception.
- Folate is the naturally occurring vitamin and occurs in 7 different forms so we have 7 different folates. Folic acid is the synthetic folate vitamin.

- Routine prescription of vitamins is not necessary as long as the pregnant woman takes a well-balanced diet. However, both iron and folic acid must be supplemented.
- To get 3–6 mg of iron daily, the diet should contain 30–60 mg of iron as only 10% is absorbed from the diet. The normal diet contains only 15 mg of iron.

B) Sleep

Eight hours at night and 2 hours rest in the afternoon.

C) Exercises

The usual activities and home work are allowed. Violent exercises are avoided. In the last month, the woman is asked to walk for an hour daily to help engagement of the head.

D) Travelling

Safe during the first half of pregnancy, but it is restricted in later months. It is not allowed if there is a tendency to abortion or preterm labour. Air travel is better than train or car for long distances.

E) Coitus

It is avoided if there is tendency to abortion or preterm labour.

F) Clothing

Should be loose. Avoid high heels which increase lumbar lordosis, and backache, and increase the risk of falling as well as ankle injury.

G) Care of the Teeth

Regular cleansing and if necessary a dentist is consulted.

H) Breasts

Crusts or dried secretion over the nipples are washed by warm water and soap or boric lotion. The nipples are drawn for a short time daily by the thumb and finger and painted with a lubricant during the last 6 weeks.

I) Bowels

Constipation is avoided by vegetables, milk, regulation of the bowels, exercises and laxatives if necessary. Avoid the use of liquid paraffin for a long time as it interferes with absorption of fat soluble vitamins A and D.

J) Bathing

The bath is given by shower. Tub and sea bathing are avoided for fear of ascending infection.

FREQUENCY OF EXAMINATION

The pregnant woman is examined every month until the 28th week; then every 2 weeks until the 36th week; and then weekly until delivery. More frequent examinations are necessary in high risk pregnancies. In the first visit the history is taken, general, abdominal and vaginal examinations are performed. Routine special investigations mentioned before are carried out. In the return visits the patient is asked about any complaint. The blood pressure and weight are recorded. Abdominal examination is performed and oedema is looked for. Urine is tested for protein and sugar. At 36th week, internal pelvimetry is done and we test for cephalopelvic proportion. A screening test to diagnose gestational diabetes is done between 24–28 weeks. Pelvic sonography is ordered routinely in the first trimester and between 16 and 18 weeks or later to diagnose fetal anomalies.

SONOGRAPHY

It means the use of sound waves with frequency above the range of human hearing hence the term ultrasonography. The frequency of audible sound waves lies between 20 and 20000 Hertz (Hz). Hertz is the standard unit of frequency and equals one cycle or wave per second. For diagnostic purposes the frequency of sound waves ranges between 1 and 30 Megahertz (MHz). The Megahertz equals one million Hertz.

The ultrasound machine contains crystals which change electric waves into sound waves and vice versa. When the electric current is applied to the machine, the crystals generate sound waves which are transmitted through the body. These sound waves are reflected from the tissues. The reflected echoes are converted by the machine to electric waves and form a sound (Doppler ultrasound) or a picture of the area examined (sonography).

Ultrasound waves can pass through fluids and solid structures but can not pass effectively through gases, and so a gel must be applied between the transducer (probe) of the ultrasound machine and skin. The transducer is a device capable of converting energy from one form to another. In ultrasonography, the transducer consists of the crystal and surrounding housing.

For transabdominal ultrasound, the urinary bladder must be full to allow obstetric examination in the first trimester of pregnancy and also for gynaecological diagnosis.

For transvaginal ultrasound, a full bladder is not needed. Transvaginal ultrasound is more sensitive for diagnosis of tubal and ovarian masses, as the vaginal probe is nearer to the structures to be examined. It also diagnoses the pregnancy sac one week earlier than transabdominal ultrasound (at about 4 weeks and 5 weeks respectively).

INDICATIONS IN OBSTETRICS

A) Diagnostic

Sonography is used for the diagnosis of: (1) pregnancy; (2) ectopic pregnancy; (3) hydatidiform mole; (4) missed abortion, fetal viability is diagnosed by detecting movement of fetal heart and fetal limbs at 6 weeks; (5) intrauterine death; (6) incompetent cervix; (7) multiple pregnancy; (8) fetal malformation as anencephaly, hydrocephalus, cardiac and renal anomalies, etc. Routine screening by ultrasound at 16–18 weeks gestation or later can detect most of fetal abnormalities; (9) hydrops fetalis; (10) position and presentation of the fetus; (11) polyhydramnios and oligohydramnios; (12) fetal age; (13) fetal weight; (14) intrauterine growth restriction by measuring certain parameters; (15) fetal well-being by performing the nonstress test, the contraction stress test, and biophysical profile; (16) placenta praevia; (17) maturation of the placenta; (18) retained

placental fragments in postabortive and postpartum haemorrhage; (19) missed intrauterine device; (20) associated tumours as fibroids and ovarian cysts; (21) maternal diseases as hydronephrosis, and gallbladder stones; (22) the state of caesarean section scar. If the thickness of the scar after 36 weeks of pregnancy is 4.5 mm or more, rupture of the scar is not liable to occur if vaginal delivery is allowed.

Sonography is done routinely at 10–12 weeks to confirm gestational age, to diagnose missed abortion, ectopic pregnancy, multiple pregnancy, uterine and adnexal pathology and to screen for Down syndrome.

B) Invasive Procedures

The following invasive procedures (diagnostic and therapeutic) are done under ultrasound guide: (1) chorionic villus biopsy; (2) amniocentesis; (3) cordocentesis; (4) taking biopsy from fetal skin and liver; (5) intrauterine fetal transfusion in case of erythroblastosis fetalis; (6) fetal shunt operations as in case of hydrocephalus, and bilateral hydronephrosis (see fetal therapy); (7) salpingocentesis in the undisturbed tubal pregnancy; (8) selective embryo reduction (see multiple pregnancy).

DIAGNOSIS OF GESTATIONAL AGE

1. In first trimester. We measure the volume of the pregnancy sac and the crown-rump length. The latter is measured between 6 and 12 weeks of pregnancy. It is not measured after the 12th week as the fetus becomes curved.
2. In second trimester. We measure the biparietal diameter, head circumference, abdominal circumference, and femur length.

HAZARDS OF SONOGRAPHY

Available data indicate that sonography is safe for the mother and fetus. The procedure has replaced X-ray examination in obstetrics to avoid hazards of irradiation. X-ray diagnosis is resorted to if ultrasound is not available.

HAZARDS OF IRRADIATION

(1) Fetal malformation; (2) fetal death; (3) affection of the gonads of the mother and fetus leading to abnormal mutations in the genes of sex cells and this may be harmful to the offspring causing infertility, abortion, or congenital malformation; (4) increased risk of fetal malignant disease as leukemia during the early years of life. The risk is one in 5000 exposed fetuses.

Exposure to 5 rads or more during pregnancy causes harm to the fetus. A single abdominal X-ray exposes the fetus to 0.5 rad, pelvimetry to 1 rad.

Note. The least mature placenta is grade 0, and the most mature is grade 3 which shows increased deposition of calcium within the placental septa. Grade 3 is associated with a mature L/S ratio.

DOPPLER ULTRASOUND

Doppler is the name of an Austrian scientist. Doppler ultrasound is used to measure blood flow and velocity in blood vessels. The ultrasound waves are reflected from moving red blood cells (RBCs) in the blood vessel. The returning echoes have a frequency related to the velocity of RBCs. In obstetrics, Doppler ultrasound is used to measure blood flow in maternal blood vessels including the uterine artery and arcuate arteries in the placental bed, as well as fetal blood vessels including the umbilical artery, descending aorta, internal carotid artery, and cerebral arteries as the middle cerebral artery (to have idea about cerebral blood flow). Doppler ultrasound can show increased vascular resistance and reduced blood flow in the fetoplacental circulation in cases of placental insufficiency as pre-eclampsia and intrauterine growth restriction. In such cases the diastolic blood flow in the umbilical artery may be reduced, absent, or even reversed (blood flows from placenta towards the fetus).

INDICES TO MEASURE BLOOD FLOW

1. Systolic/diastolic (S/D or A/B) ratio. The peak of systolic pressure is divided by end-diastolic pressure, i.e., $\frac{S}{D}$.
2. Resistance (Pourcelot) index = $\frac{S-D}{S}$.
3. Pulsatility index = $\frac{S-D}{\text{Mean}}$.

An increase in any of the above indices indicates a decrease in the blood flow and vice versa.

Doppler ultrasound is also used to auscultate the fetal heart sounds using the Doppler stethoscope as the Sonicaid or Doptone. It is also used for continuous recording of the fetal heart to show abnormal patterns during performing the nonstress test or contraction stress test.

ABNORMAL PREGNANCY

HAEMORRHAGE IN EARLY PREGNANCY

(First 20 Weeks)

CAUSES

(1) Abortion; (2) ectopic pregnancy; (3) hydatidiform mole; (4) local gynaecological lesions as cervical erosion or carcinoma; (5) bleeding may occur at the time of menstruation during the first three months of pregnancy. It is due to separation of parts of the decidua not occupied by the ovum. This can occur because the decidua capsularis and decidua parietalis do not fuse except at the 12th week.

ABORTION

DEFINITION

It is expulsion or removal of the products of conception from the uterus before viability of the fetus, that is before 20 weeks of pregnancy or if fetus weighs less than 500 g or its length is less than 25 cm (crown-heel length). Viability starts after the twentieth week and means that when the fetus is expelled from the uterus it can survive under favourable conditions.

INCIDENCE

About 20% of all pregnancies end in abortion. Most cases (80%) occur in the third month (8–12 weeks) because at this period the level of progesterone may drop as the corpus luteum is degenerating and the placenta is not yet completely formed to carry out its function.

AETIOLOGY

Abortion may be accidental or habitual (recurrent).

Causes of Accidental Abortion

1. Chromosomal abnormalities of the fetus.
2. Trauma as blow on the abdomen or operative trauma.
3. Fevers.
4. Maternal hypoxia as in badly given anaesthesia.
5. Overdistension of the uterus by multiple pregnancy or acute polyhydramnios.
6. Psychological disturbances.
7. Abortifacient drugs as quinine.

Causes of Habitual Abortion: will be discussed later on.

MECHANISM OF ABORTION

Before the 12th week the pregnancy sac tends to be expelled from the uterus in one mass. After that time the process is similar to labour in that the membranes rupture with escape of amniotic fluid then the fetus and placenta are born separately

CLINICAL TYPES OF ABORTION

Abortion may be: (1) Threatened; (2) inevitable; (3) incomplete; (4) complete; (5) missed; (6) septic; (7) cervical; (8) therapeutic; (9) criminal and (10) habitual.

Threatened Abortion

Diagnosis

1. Symptoms and signs of pregnancy.
2. Bleeding, which is usually slight or moderate. It is due to separation of the ovum from the uterine wall.
3. Pain is absent or heaviness may be felt in the suprapubic region.
4. On examination the uterus is found enlarged and the internal Os of the cervix is closed.
5. Pregnancy test is positive.
6. Sonography will show a viable fetus and differentiates threatened abortion from missed abortion and hydatidiform mole.

End Result of Threatened Abortion

1. Bleeding stops, the embryo or fetus is still alive and pregnancy continues (50%); or:
2. Bleeding stops, the embryo or fetus dies but is retained in the uterus leading to missed abortion; or:
3. Bleeding increases, uterine contractions occur, and cervix dilates leading to inevitable abortion.

Treatment

1. Rest in bed until the bleeding stops (red loss) and for some days afterwards.
2. Travelling and sexual intercourse are avoided.
3. Sedatives to relieve pain and anxiety of the patient, e.g. diazepam (Valium) 5 mg orally tds.
4. Treatment of the cause, e.g. hypertension.
5. Hormones: Thyroxine is given in case of hypothyroidism. Progesterone is of no value in the treatment of threatened abortion even if there is evidence of hormone deficiency.
6. Evacuation of the uterus is indicated if severe bleeding occurs at any time.

Inevitable Abortion

Diagnosis

1. Symptoms and signs of pregnancy.
2. Bleeding, which is usually severe.
3. Pain: It is colicky intermittent felt in the suprapubic region (uterine contractions) and accompanied with low backache (cervical dilatation).
4. On examination, the uterus is enlarged and the internal Os of the cervix is dilated. The products of conception may be felt through the dilated cervix. In this case, abortion will occur in spite of any treatment.

Treatment

Vaginal evacuation and curettage or suction evacuation if duration of pregnancy is less than 12 weeks.

After 12 weeks oxytocin is given by intravenous drip to help the uterus expel its contents. If the placenta is retained it has to be removed under general anaesthesia followed by curettage of the uterine cavity.

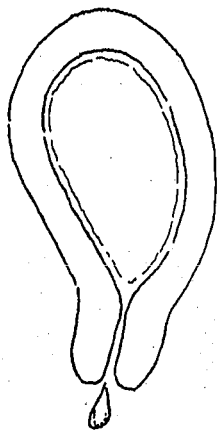


Fig. 21. Threatened abortion

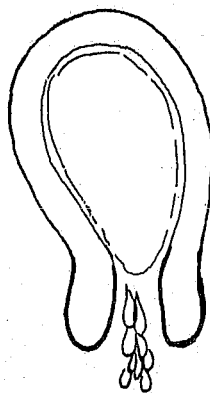


Fig. 22. Inevitable abortion

Incomplete Abortion

Part of the products of conception is expelled from the uterus and part is retained inside the uterus. The clinical picture is like inevitable abortion so there is bleeding, colicky pain, the internal Os is dilated and the retained product may be felt through it. Treatment is like inevitable abortion.

Complete Abortion

All the products of conception are expelled from uterus. The bleeding is slight and gradually diminishes, pain is absent, the uterus is smaller than the period of amenorrhoea and the cervix is closed or closing. Ultrasound is performed. If the uterus is empty nothing is done. If remains are seen, evacuation is carried out.

N.B.: In all cases of abortion the products of conception must be examined macroscopically and microscopically.

Missed Abortion

Definition

The dead products of conception are retained inside the uterus.

Carneous mole is a special type of missed abortion which is due to repeated attacks of haemorrhage occurring into the choriodecidual space around the ovum before the 12th week of pregnancy. The ovum becomes surrounded by blood coagulated in layers forming a bloody or fleshy mole.

Diagnosis

A. Symptoms

1. Symptoms of threatened abortion may or may not occur.
2. Pregnancy symptoms gradually disappear as nausea, vomiting and breast symptoms.
3. Failure of the abdomen to increase in size.
4. Failure to feel fetal movements or cessation of fetal movements if previously present.
5. Milk secretion may start spontaneously from the breasts (La Monte du Lait), frequent in second trimester abortion and due to drop in secretion of oestrogen which normally blocks the action of prolactin on the breast.
6. A dark brown vaginal discharge may occur (prune juice discharge).

B. Signs

1. The uterus is smaller than the period of amenorrhoea and fails to enlarge. It is firm in consistency.
2. Fetal heart sounds (FHS) cannot be heard by Sonicaid.

C. Investigations

1. Sonography is diagnostic. It shows a collapsed pregnancy sac or absent fetal heart movements.
2. Pregnancy test becomes negative within two weeks after death of the embryo or fetus, sometimes remains positive for a longer period if there is still living chorionic tissue.

Complications

1. Intrauterine infection
2. Disseminated intravascular coagulation (DIC) and hypofibrinogenaemia. In few cases of missed abortion, the degenerated ~~placenta~~ or dead-fetus liberates thromboplastin which enters the maternal circulation causing intravascular clotting and hypofibrinogenaemia. This leads to maternal haemorrhage in the form of epistaxis, haematemesis and uterine bleeding at the time of abortion because the blood fails to coagulate. The condition is liable to occur (in about 25% of cases) if the duration of pregnancy is more than 16 weeks and the dead fetus is retained more than one month.

Treatment

If the size of the uterus is less than 12 weeks vaginal or suction evacuation is done. If the size of the uterus is more than 12 weeks we wait 4 weeks so that the uterus may expel its contents spontaneously (it does so in 90% of cases), if this fails we induce abortion by:

1. Oxytocin infusion (up to 50 units or more in 500 ml 5% glucose in normal saline) or
2. Prostaglandins and prostaglandin analogues (the compounds can be given orally, intramuscularly, intravenously, intra-amniotic, extra-amniotic or in the form of vaginal suppositories or gel).

If these methods fail we can try intra-amniotic injection of hypertonic saline solution 20% or urea solution 40% in 5% glucose or do abdominal hysterotomy.

N.B.:

1. Immediate interference in missed abortion is indicated if there is infection, bleeding, or if the patient is anxious.
2. Blood is tested for its fibrinogen content before the induction of abortion.

Septic Abortion

It is any type of abortion complicated by infection. It is usually the result of criminal interference.

Causative Organisms

Any pathogenic organism can cause the infection. Organisms include staphylococci, streptococci, *Escherichia (E.) coli*, *clostridium welchii* as well as other organisms. The commonest organisms are the anaerobic streptococci.

Routes of Infection

1. *Endogenous*: Infection by organisms already present in the genital tract, e.g. anaerobic streptococci.
2. *Exogenous*: Organisms are transmitted to the genital tract from an external source such as instruments, dust, flies and droplet infection.

3. *Autogenous*: Organisms are transmitted to the genital tract from a septic focus in the patient. This occurs by the bloodstream or by fingers of the patient.

Clinical Picture

1. *General manifestations of infection* in the form of fever, rapid pulse, headache and vomiting. If the pulse rate is more than 110 beats per minute it usually indicates spread of infection beyond the uterus. In case of septicaemia the pulse rate is out of proportion to the rise of temperature. Infection with *clostridium welchii* leads to haemolysis of red cells, severe anaemia and jaundice. Bacteraemic or septic shock leads to hypotension.
2. *Abdominally*; there is suprapubic pain, tenderness and may be rigidity.
3. *Vaginally*; there is an offensive discharge.

Investigations

1. A cervicovaginal swab is taken from cervical canal and vaginal vault for culture (aerobic and anaerobic) and sensitivity test.
2. Blood for culture (aerobic and anaerobic) and sensitivity test (if pyrexia is 39° C or more).
3. Complete blood count (CBC).
4. Urine analysis.
5. Kidney function tests: Blood urea, serum creatinine and serum electrolytes. The most serious complication of septic shock or *clostridium welchii* infection is renal failure.
6. Coagulation profile if DIC develops.
7. X-ray of the abdomen in the upright position. It can show air under the diaphragm indicating perforation of uterus or vaginal vault, it diagnoses physometra and the presence of a foreign body as a catheter in the peritoneal cavity.

Treatment

1. Hospitalization, and isolation in a separate room.
2. Observation for vital signs (pulse, temperature and blood pressure), fluid intake and urine output.
3. Fluid therapy in the form of 5% glucose and normal saline or lactated Ringer solution to maintain a urinary flow of at least 30 ml per hour.
4. Antibiotics, given according to sensitivity test. We can start by giving penicillin (or ampicillin) and an aminoglycoside as gentamycin and in severe cases triple therapy by adding clindamycin (or metronidazole).
5. Anti-gas gangrene or antitetanic serum only given in infection with *Cl. welchii* or tetani.
6. Oxytocin infusion to control bleeding and help passage of retained products.
7. Packed red cells may be given to correct anaemia.

8. Symptomatic treatment in the form of antipyretics and analgesics.
9. Evacuation of the uterus is indicated if it contains products of conception. Removal of the contents is followed by sharp uterine curettage. There is no harm as long as antibiotic therapy has been started.
10. Hysterectomy is indicated in case of gangrene of the uterus or septic shock not responding to treatment.

Complications of Septic Abortion

- (1) Peritonitis; (2) septicaemia; (3) septic shock; (4) renal failure; (5) gas gangrene; (6) DIC; (7) haemolytic anaemia; (8) jaundice; (9) adult respiratory distress syndrome.

Notes:

- Septic shock is now called systemic inflammatory response syndrome.
- *Adult respiratory distress syndrome (shock lung)*. The bacterial endotoxin leads to damage of the alveolar cells and increased permeability of alveolar capillaries. This leads to interstitial lung oedema and formation of an exudate known as hyaline membrane which lines the alveoli, alveolar ducts, and respiratory bronchioles. The changes lead to respiratory insufficiency.

Cervical Abortion

The products of conception are separated from the uterus but retained inside the cervical canal because of stenosed external Os due to previous cauterization or operation on the cervix. The cervix becomes ballooned but the bleeding is usually slight. Under anaesthesia the cervix is dilated, products of conception removed and uterus curetted to remove the decidua.



Fig. 23. Incomplete abortion

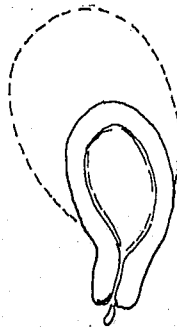


Fig. 24. Missed abortion



Fig. 25. Cervical abortion

Therapeutic Abortion

It is termination of pregnancy for a medical reason.

Criminal Abortion

It is termination of pregnancy for a non medical reason.

Note: Elective or voluntary abortion is termination of pregnancy for a non medical reason on the request of the woman in countries where abortion is legalized.

Habitual Abortion

It means three or more successive spontaneous abortions.

Incidence

Less than 1% of pregnancies (0.3–0.9%).

Actiology

The causes may be fetal, maternal, paternal, immunological or idiopathic.

I) Fetal Factor

About 50% of fetuses aborted in the first trimester show chromosomal abnormalities (commonest cause). The commonest anomaly is trisomy.

II) Maternal Factors: General or local

General Causes

1. Chronic hypertension due to any cause, e.g. essential or secondary as in renal disease. Hypertension interferes with placental circulation.
2. Endocrine disturbances :
 - a) Insufficient production of progesterone by the corpus luteum. This luteal phase defect (LPD) leads to poor secretory changes in the endometrium, thus interfering with the implantation or growth of the ovum (5–35% of cases).
 - b) Hypo- or hyperthyroidism.
 - c) Uncontrolled diabetes mellitus.
 - d) Hyperprolactinaemia.
 - e) High level of luteinizing hormone in the follicular phase of the cycle (8th day) more than 10 mIU/ml. This may produce a poor quality ovum.
3. Severe malnutrition. Deficiency of specific vitamins as C, E and folic acid do not cause abortion.
4. Chronic anaemia (fetal hypoxia).
5. Psychological disturbances may be a factor but not definitely proven.
6. Chronic maternal infection with *Toxoplasma*, *Listeria monocytogenes* and *Ureaplasma urealyticum*. However, some do not believe that chronic maternal infection is a cause of repeated abortions.
7. Smoking and alcohol (even when consumed in moderation).

8. Inherited thrombophilia. Deficiency of protein C or S or antithrombin III (natural anticoagulants). This leads to arterial or venous thrombosis including placental blood vessels. Also mutated factor 5 and hyperhomocysteinaemia.

Note: Syphilis is not considered a cause of abortion as the spirochetes cross the placenta in a significant number after the 20th week of gestation when the Langhans layer starts to disappear. Also ABO and Rh incompatibility between the husband and wife is a cause of intrauterine death but not a cause of abortion.

Local causes

1. Uterine hypoplasia.
2. Congenital malformation of the uterus as bicornuate, septate and subseptate uterus.
3. Incompetent cervix (patulous os).
4. Abnormal position of uterus. Severe retroversion or uterine prolapse.
5. Uterine endometriosis (adenomyosis) due to increased production of prostaglandin and decreased blood flow in the uterine wall.
6. Abnormal vascular anatomy of the uterus as 2 ascending branches of uterine artery on one or both sides.
7. Endometrial polypi.
8. Intrauterine adhesions (Asherman syndrome) due to lack of space for the growing ovum.
9. Pelvic tumours as fibroids and ovarian tumour.

III) Paternal Factor

Chromosomal abnormalities in the father and subsequently in the spermatozoa and fetus can cause abortion.

IV) Immunological Factors (10%)

1. The wife may produce antibodies against her husband's sperms. These antisperm antibodies may cause infertility or abortion.
2. Autoimmune diseases as systemic lupus erythematosus.
3. Antiphospholipid antibody syndrome. The presence of antiphospholipid antibodies as anticardiolipin antibodies or lupus anticoagulant. These immunoglobulins may lead to placental thrombosis and fetal loss.
4. When the husband and wife share unusual number of human leucocyte (HL) antigens the wife may abort repeatedly.

V) Idiopathic

In some cases, no cause is found to explain abortion.

Notes:

- The HL antigens are present on the outer surface of all cells in the human body except the red cells.
- High level of LH in the follicular phase may lead to early meiosis and formation of aged oocytes. Such abnormal ova may not become fertilized leading to infertility and if fertilized they tend to abort. This is seen in women with polycystic ovary syndrome who become pregnant.
- After 3 abortions the chance of a fourth abortion is 25-50%.

Diagnosis (Investigation)**A) History**

A careful history can give idea about the aetiology. Abortions in the first trimester are usually the result of fetal chromosomal anomalies. Hypoplasia of the uterus causes repeated abortions at increasing periods of gestation. In case of incompetent cervix, repeated abortions occur after 12th week of pregnancy (second trimester). Typically abortion starts by rupture of membranes followed by rapid expulsion of a normal fetus with little pain.

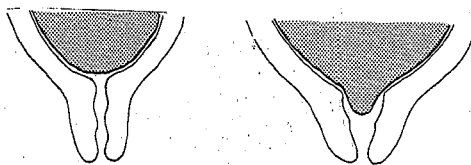


Fig. 26. Incompetent cervix provides poorer support for the bag of forewaters.

B) General Examination

This may detect a general cause as hypertension or thyroid disease.

C) Abdominal Examination

May reveal a pelvi-abdominal mass as fibroid or ovarian tumour.

D) Vaginal Examination

To exclude a local cause as uterine hypoplasia or retroversion. Incompetent cervix is diagnosed if we can pass a Hegar dilator No. 8 through the internal Os without pain or resistance. During pregnancy an abnormally short cervix or a dilated cervix with bulging membranes diagnoses incompetent cervix.

N.B.: Hegar dilator No. 8 means its diameter is 8 mm.

E.) Special Investigations

1. Urine analysis (for chronic renal disease).

2. Blood picture (for anaemia).
3. Glucose tolerance test to exclude diabetes mellitus.
4. Kidney function tests.
5. Thyroid function tests if we suspect thyroid disease.
6. Hysterosalpingography (HSG) done in all cases. It may show a uterine malformation (bicornuate uterus) a submucous fibroid, intrauterine adhesions or incompetent cervix (funnel shape appearance). The cervicogram is done in the luteal phase to exclude patulous Os. Unopposed oestrogen contracts the uterine isthmus.
7. Hysteroscopy alternative to HSG to diagnose uterine abnormalities.
8. Sonography may show uterine anomalies and during pregnancy may diagnose incompetent cervix.
9. Serum progesterone to diagnose luteal phase deficiency (on day 22-24 of the cycle).
10. Serum level of luteinizing hormone in the follicular phase of the cycle (8th day).
11. Cervical culture for *Listeria monocytogenes*.
12. Endometrial culture for *ureaplasma urealyticum*.
13. Serological test for toxoplasmosis.
14. Study of chromosomes in husband and wife (5% will show chromosomal abnormalities).
15. Maternal serum for antisperm antibodies.
16. Check for systemic lupus erythematosus.
17. Test maternal serum for lupus anticoagulant and anticardiolipin antibodies.
18. Investigation of husband and wife for HL antigens or maternal serum is tested for blocking antibodies.

Note: All physicians investigate the case after 2 successive spontaneous abortions and not 3.

Treatment

It is treatment of the cause as hypertension or hypothyroidism. Uterine fibroids are removed by myomectomy. Incompetent cervix is treated by Shirodkar or McDonald operation. It is done during pregnancy after the 12th week after confirming fetal viability by ultrasound. The best time is between 12 and 14 weeks. In Shirodkar operation a non absorbable material as nylon thread or Mersilene (Dacron) tape is passed around the cervix deep to the mucosa at the level of the internal os. The suture is removed 2 weeks before full term to allow for vaginal delivery. In McDonald operation, four *bites* are taken to surround the cervix as high as possible and the suture is tied. The suture is tied anteriorly or posteriorly, but usually anteriorly to allow easy removal. It is easier than Shirodkar operation and gives the same success rate (85-90%). Transabdominal cervical cerclage may be indicated when the cervix is very short, amputated (as after Fothergill

operation), deeply lacerated, congenitally deformed, after cervical conization, and after repeated failure of vaginal cerclage. Operation is also done after the 12th week. The patient is delivered by caesarean section at 38 weeks and the ligature is left in place to allow more pregnancies.

If there is no apparent cause, i.e., idiopathic habitual abortion the treatment is empirical and consists of:

1. Measures before pregnancy: The patient should wait 3 months after abortion before starting a new pregnancy with correction of anaemia and general condition. The couple are advised to stop smoking and alcohol.
2. Measures after pregnancy:
 - More periods of rest.
 - Travelling and intercourse are avoided for several weeks during the critical phase when abortion used to take place.
 - Sedatives.
 - Progesterone may be given empirically to improve placentation, to prevent expulsive uterine contractions, to increase the tone of the cervical isthmus, and to inhibit the maternal immune response which tends to expel the conceptus.
 - Psychotherapy if necessary.

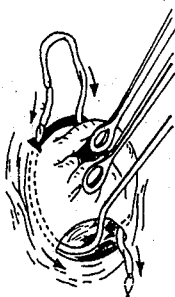


Fig. 27. Shirodkar operation

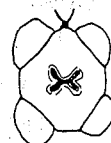
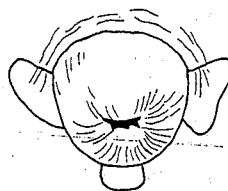


Fig.28. McDonald operation

INCOMPETENT CERVIX

It leads to painless (silent) dilatation of the cervix causing second trimester abortion or preterm labour.

Incidence

About 1 in 500 pregnancies. However, the actual incidence is unknown due to difficult objective diagnosis.

Aetiology

1. Congenital weakness.
2. Traumatic. This may follow operations on the cervix as dilatation and curettage, forceps or breech extraction before cervix is fully dilated. Trauma is the commonest cause.
3. Dysfunctional. Due to lack of progesterone. Progesterone causes contraction of the isthmus while oestrogen causes relaxation. Also a high level of relaxin hormone may be responsible.
4. Exposure to diethylstilbesterol in utero. The drug is no more used in pregnancy.

Diagnosis

- A. History. Typical history mentioned before.
- B. Investigations done in absence of pregnancy:
 1. The use of Hegar dilator. See before.
 2. Uterine sound. When the internal Os is wide the sound rocks freely inside it, and there is absence of a closing snap.
 3. The use of a pediatric Foley catheter. A catheter with a 3-ml bulb is introduced into the uterine cavity. The bulb is filled with 3 ml saline. In case of incompetent cervix, the catheter is withdrawn through the internal Os without resistance.
 4. Hysterosalpingography. It shows a funnel-shaped cervix.
- C. Investigations done during pregnancy:
 1. Serial cervical assessment by digital examination carried out weekly between 14 and 26 weeks of pregnancy. An abnormally short cervix, or a dilated cervix with bulging membranes, diagnoses cervical incompetence.
 2. Serial vaginal sonography during pregnancy done between 14 and 26 weeks. Incompetence is diagnosed if the cervix is short and less than 2.5 cm, or internal Os is dilated more than 1.5 cm in the first trimester, and more than 2 cm in the second trimester. If the cervix looks normal, the patient is asked to bear down or pressure on the uterine fundus is applied. In this case funneling of the cervix diagnoses incompetence.

Treatment

Cervical cerclage is done during pregnancy after the 12th week and after confirming fetal viability by ultrasound. The best time is between 12 and 14 weeks. After that time it will be difficult to put the ligature at the level of the internal cervical Os. Operation is done transvaginally or transabdominally as mentioned before.

POSTABORTIVE BLEEDING

It is persistent or recurrent bleeding within the first 4 weeks after abortion.

Causes

1. Incomplete abortion with retained products of conception.
2. Uterine infection due to separation of a septic slough. Bleeding may come from placental site or cervix.
3. Perforation of uterus or trauma to the cervix.
4. Submucous fibroid or fibroid polyp due to necrosis and infection.
5. Malignant trophoblastic disease.
6. Local gynaecological lesion as cervical erosion and carcinoma of cervix.
7. Haemorrhagic blood disease as thrombocytopenia.
8. Dysfunctional uterine bleeding.

Notes for the Postgraduate

1. Following complete abortion, ovulation occurs after 2–3 weeks and menstruation after 4–6 weeks.
2. It is suggested that after implantation the foreign paternal antigens in the trophoblast stimulate the mother immune system to produce blocking antibodies which will coat the fetal antigens and prevent the fetus from being rejected by the maternal T-lymphocytes. If the husband and wife share unusual number of HL antigens (3 or more or the Dr antigen alone) the trophoblast may fail to stimulate the production of blocking antibodies and the pregnancy is rejected. In this case the wife is immunized by injecting paternal lymphocytes intradermally, subcutaneously and intravenously as well. In this way the wife will produce the blocking antibodies (antipaternal cytotoxic antibodies) and abortion will not occur.
3. The lupus anticoagulant is an immunoglobulin found in women with lupus erythematosus (5–15%) and other connective tissue diseases as well as in women without systemic disease. It leads to thrombosis in the placental circulation and pregnancy loss. The anticardiolipin antibodies have the same effect. The condition is treated by low-dose aspirin 75 mg daily combined with heparin throughout pregnancy (5000 units subcutaneously twice daily or low-molecular weight heparin once or twice daily, and its dose depends upon the preparation). If this therapy fails, immunoglobulin infusion is given (IgG).

ECTOPIC PREGNANCY**DEFINITION**

Pregnancy in abnormal site that is outside the normal uterine cavity.

TYPES

(1) Tubal; (2) ovarian; (3) abdominal or intraperitoneal; (4) intraligamentary (between the 2 layers of the broad ligament); (5) cervical; (6) angular pregnancy in which the fertilized ovum is implanted in the angle of the uterus; (7) pregnancy in a rudimentary horn. The commonest site of ectopic pregnancy is the fallopian tube (over 95%).

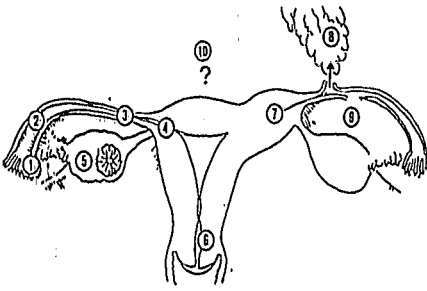


Fig. 29. Sites of ectopic pregnancy.

(1) Fimbrial, (2) Ampullary, (3) Isthmic, (4) Interstitial, (5) Ovarian, (6) Cervical, (7) Rudimentary horn, (8) Secondary abdominal pregnancy, (9) Broad ligament, (10) Primary abdominal.

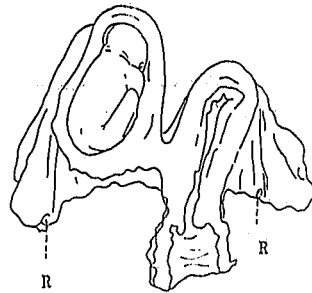


Fig. 30. Pregnancy in a rudimentary horn. R. Round ligament.

TUBAL PREGNANCY

Incidence

It varies between 1 in 80 to 1 in 200 pregnancies; average 1%.

Aetiology

I) Causes in the tube:

- a) Mechanical factors interfering with the passage of the fertilized ovum. These may be:
 1. Congenital: As long and narrow tube, diverticulae and accessory ostia.
 2. Traumatic: Following operation on the tube as salpingoplasty and tubal ligation.
 3. Inflammatory: Chronic salpingitis is the commonest cause of tubal pregnancy (50%). Inflammation leads to stenosis of the tube, destruction of cilia, etc.
 4. Neoplastic: Narrowing of the tube by a fibroid or a broad ligament tumour.
 5. Functional: As tubal spasm or antiperistaltic contractions.
- b) The presence of areas of endometriosis in the tube. This encourages embedding of the fertilized ovum.
- c) Change in tubal motility caused by exogenous hormones. The incidence is increased in women receiving progestin-only pills and drugs which induce ovulation as Clomid and gonadotrophins. Progesterone reduces tubal motility and ciliary movements.
- d) Smoking at the time of conception which reduces tubal motility and ciliary movements.

II) Causes in the ovum:

- a) Rapid development of the chorionic villi.
- b) Rapid disappearance of the corona radiata and zona pellucida.
- c) Transperitoneal migration of the ovum from the opposite ovary, so when it reaches the opposite tube it is too large to pass through it. Tubal pregnancy is found on the opposite site of the corpus luteum in 20% of cases.

III) The intrauterine contraceptive device (IUD) increases the incidence of ectopic pregnancy by changing tubal motility, by causing salpingitis and by preventing intrauterine pregnancy but not tubal pregnancy leading to a relative increase in the rate of tubal pregnancy. When pregnancy occurs in the presence of IUD the rate of ectopic is 2-3%.

Pathology

1) Ovaries

One ovary contains the corpus luteum of pregnancy. The presence of 2 corpora lutea indicates associated intra- and extrauterine pregnancy.

2) Uterus

It is slightly symmetrically enlarged and soft in consistency reaching about 8 weeks pregnancy. The endometrium shows a decidual reaction. These changes are caused by the hormones of pregnancy oestrogen and progesterone. When the ectopic pregnancy becomes disturbed there is a drop in the level of human chorionic gonadotrophin (HCG), regression of the corpus luteum, drop in the level of oestrogen and progesterone leading to separation of the decidua and vaginal bleeding. The decidua comes out in small pieces or it is expelled as an intact uterine cast triangular in shape. Sometimes the endometrium shows the Arias-Stella reaction (10-15% of cases).

3) Tube

The ovum may be implanted in any part of the tube, however, the commonest site is the ampulla. Because the decidual reaction in the tube is poor the ovum penetrates deeply and becomes embedded in the muscle layer. The chorionic villi erode the blood vessels in the wall leading to haemorrhage around the ovum followed by internal or external tubal rupture.

i) Internal Tubal Rupture

It is rupture through the mucosa, bleeding occurs inside the tube, the results are:

a) Tubal Mole

The blood coagulates in layers around the ovum in the lumen of the tube forming a fleshy mass or mole (missed abortion). This may become absorbed or infected forming a pyosalpinx.

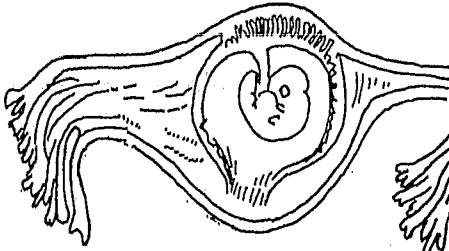


Fig. 31. Internal tubal rupture

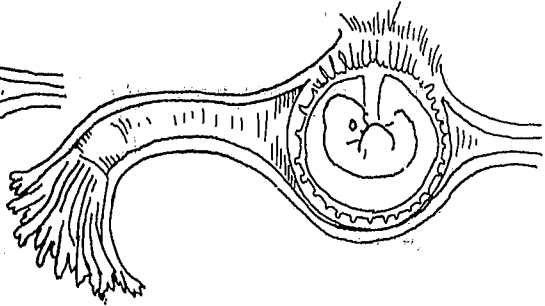


Fig. 32. External tubal rupture

b) Tubal Abortion

When the ovum reaches the lumen of the tube it becomes expelled by tubal contractions through the abdominal ostium. Abortion is complete when the ovum reaches the peritoneal cavity and incomplete when it remains at the abdominal ostium. The blood from the site of rupture may be slight and collects around the fimbrial end of the tube forming a peritubal haematoma or haematocele or it may be moderate and accumulates gradually into Douglas pouch forming a pelvic haematocele. Sometimes the bleeding is excessive, leading to severe intraperitoneal haemorrhage.

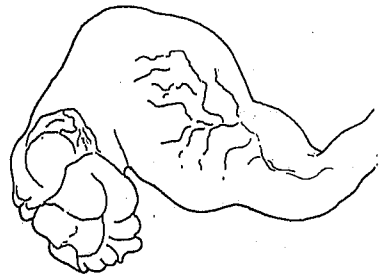


Fig. 33. Tubal abortion

ii) External Tubal Rupture

Bleeding occurs outside the tube. The rupture may be intraperitoneal or extra-peritoneal:

a) Intraperitoneal Rupture

Rupture through the roof and sides of the tube. It may lead to:

1. Paratubal haematocele: Blood collects along the side of the tube.
2. Pelvic haematocele.
3. Severe intraperitoneal haemorrhage, or
4. Secondary abdominal pregnancy.

b) Extraperitoneal Rupture

Rupture through the floor of the tube. It may lead to broad ligament haematoma with death of the ovum or intraligamentary pregnancy.

Fate of the Ovum

In the great majority of cases the ovum dies after tubal rupture and is absorbed. In few cases the pregnancy sac remains intact and the placenta grows out of the tear to gain a new blood supply from the surrounding organs, e.g. omentum or intestine and forms a secondary abdominal pregnancy.

Fate of Abdominal Pregnancy

1. In the great majority of cases (90%) the fetus dies because of poor blood supply and may become mummified or calcified forming a lithopaedion which may remain for many years without symptoms.
2. Sometimes the pregnancy sac becomes infected from the bowel or bloodstream forming an abscess. The abscess may open into the bladder, vagina, rectum or abdominal wall with discharge of pus and fetal parts.
3. In few cases the fetus grows to full term when for unknown reason spurious or false labour occurs which is followed by rupture of the sac with intraperitoneal haemorrhage or death of the fetus. Such fetus is usually malformed (50%) due to poor blood supply.

Diagnosis

1) Undisturbed Tubal Pregnancy (2%)

It may be discovered accidentally during the routine examination of the pregnant woman.

Symptoms

1. There is a short period of amenorrhoea for 1 or 2 months.
2. Symptoms of early pregnancy as nausea or vomiting.
3. A dull aching pain is usually present in one iliac fossa. It is due to distension of the tube and stretching of its peritoneal coat.

Signs

1. *General examination:* Breast signs of pregnancy.
2. *Abdominal examination:* Tenderness in one iliac fossa.
3. *Vaginal examination:* Local signs of pregnancy. The cervix is soft and severe pain occurs when it is moved from side to side (jumping sign – cervical excitation). The uterus is soft and slightly enlarged. One fornix is very tender and paracervical pulsations may be felt. A mass may be felt to one side of the uterus. It is very tender, soft and may be pulsating. The posterior fornix is usually tender.

Differential Diagnosis

- 1) Tubal inflammatory mass, e.g. pyosalpinx.
- 2) Appendicular mass.

II) Subacute Type (60%)

The commonest type. It is caused by tubal mole, tubal abortion, peritubal or paratubal haematoma.

Symptoms

1. There is a short period of amenorrhoea for 1 or 2 months. Sometimes (25%) there is no history of amenorrhoea due to occurrence of postconceptional menstruation, also the woman may mistake the uterine bleeding which occurs with tubal pregnancy for a true menstrual period.
2. Symptoms of early pregnancy.
3. Pain: It is felt in one iliac fossa. It may be dull aching due to distension of the tube sharp stabbing due to repeated haemorrhage in the wall of the tube or colicky caused by tubal contractions in case of tubal abortion.
4. Fainting attacks or even shock, due to pain and irritation of the peritoneum by repeated attacks of bleeding.
5. Vaginal bleeding. It occurs after pain and is caused by separation of the decidua. It is usually slight and dark brown in colour (prune juice). Decidual fragments or even a complete cast may be noticed in the discharge. Bleeding is absent in 15%.

Signs

1. *General examination:* Varying degree of anaemia depending upon the amount of blood loss. Breast signs of pregnancy. The pulse is usually rapid, especially after an attack of pain. Temperature may be slightly raised up to 38°C due to absorption of blood from peritoneal cavity. Blood pressure falls in proportion to the amount of internal haemorrhage.
2. *Abdominal examination:* Tenderness and rigidity in one iliac fossa. Cullen sign may be present (bluish colouration around the umbilicus due to the presence of blood in the peritoneal cavity and its absorption by lymphatics). It is a late sign.
3. *Vaginal examination:* as before.

Differential Diagnosis

1. Tubal inflammatory mass, e.g. pyosalpinx.
2. Appendicular mass.
3. Uterine abortion.
4. Bleeding into a corpus luteum (haemorrhagic corpus luteum).

III) Acute Type

It is due to sudden severe intraperitoneal haemorrhage.

Symptoms

There is a sudden severe abdominal pain occurring after a short period of amenorrhoea. Shoulder pain may be felt on lying down due to diaphragmatic irritation by blood.

Signs

1. *General examination:* Evidence of internal haemorrhage.
2. *Abdominal examination:* Generalized tenderness and rigidity. There may be shifting dullness and bluish colouration around the umbilicus.
3. *Vaginal examination:* It reveals little information because of marked tenderness and rigidity so the pelvic organs cannot be felt.

Differential diagnosis

1. Causes of internal haemorrhage as rupture of the spleen.
2. Causes of acute abdominal pain as acute appendicitis. Disturbed ectopic pregnancy is the commonest cause of acute abdominal pain in a woman in the childbearing period even in absence of amenorrhoea.

IV) Chronic Type with Pelvic Haematocoele

Symptoms

1. Recurrent attacks of lower abdominal pain, fainting and vaginal bleeding after a period of amenorrhoea (a history suggestive of disturbed ectopic).
2. Pressure symptoms due to accumulation of blood in Douglas pouch as dysuria, dyschezia, and dyspareunia (bathroom sign).

Signs

1. *General:* As subacute type.
2. *Abdominal:* A pelvi-abdominal swelling may be felt which is fixed and ill-defined.
3. *Vaginal:* There is a fixed, tender mass bulging through the posterior fornix. It is soft, firm or hard if calcified and this depends on its duration. The uterus is pushed forwards or upwards by the mass. It is slightly enlarged and soft. Aspiration of Douglas pouch may reveal non-clottable blood confirming the diagnosis (clottable blood means that the needle has pierced a blood vessel).

Differential Diagnosis

1. Pregnancy with an ovarian cyst or posterior wall fibroid impacted in Douglas pouch.
2. Incarcerated retroverted gravid uterus.

V) Advanced Abdominal Pregnancy

Incidence: About 1:15,000 pregnancies.

Diagnosis

1. **History:** There is usually a history of recurrent attacks of lower abdominal pain, fainting and vaginal bleeding after a period of amenorrhoea (a history suggestive of disturbed ectopic).
2. **Abdominal Examination:** The lie of the fetus is usually abnormal, e.g. a high transverse lie. The fetal parts are easily felt. Uterine contractions cannot be detected over the mass. The fetus cannot be moved towards the midline.
3. **Vaginal Examination:** The uterus is felt separate from the mass. It is slightly enlarged and soft (orange size).
4. **Sonography.** (a) Fetus is seen separate from the uterus; (b) no uterine wall is seen between fetus and urinary bladder; (c) abnormal fetal attitude; (d) fetal parts are close to maternal abdominal wall; (e) placenta is seen outside the uterus.
5. **Computed tomography.** It confirms the diagnosis if sonography is not conclusive.
6. **Magnetic resonance imaging.** Very accurate for diagnosis.

Differential Diagnosis

Some cases of rupture of uterus.

Special Investigations in Ectopic Pregnancy

1. **Complete blood count (CBC).** Falling level of haemoglobin and haematocrit value indicates internal haemorrhage. The leucocytic count is normal in 50% of cases and elevated up to 30000/cu.mm in 50%.
2. **Urinalysis** (excludes pyelonephritis and renal colic).
3. **Urinary pregnancy tests.** The slide agglutination test using latex particles is positive in about 50–60% of cases. Sensitive urinary tests as ELISA tests (enzyme-linked immunoabsorbent assays) are positive in 90–95% of cases (can detect small amounts of HCG, 10–50 mIU/ml). However a negative urinary test does not exclude ectopic pregnancy.
4. **Radioimmunoassay** of beta-subunit of HCG in serum is a very sensitive test and detects small amounts of the hormone (1 mIU/ml). The test is positive in all cases.
5. **Serum progesterone.** A level of 25 nanograms/ml or higher indicates a viable intrauterine pregnancy. A lower level indicates a nonviable intrauterine or ectopic pregnancy.
6. **Sonography.**
 - a. Vaginal sonography is more sensitive than abdominal sonography to diagnose ectopic pregnancy. Vaginal ultrasound diagnoses the case in more than 90% of cases by showing a pregnancy sac outside the uterus.

- b. Colour Doppler ultrasound. It shows a vascular colour in a characteristic placental shape (ring-of-fire pattern) and shows a characteristic uterine and adnexal blood flow pattern (a high velocity low-impedance flow pattern compatible with placental perfusion).
7. *Combining beta HCG and ultrasound: the discriminatory zone.* When beta HCG value is above 6000 mIU/ml and no pregnancy sac is seen inside the uterus by abdominal ultrasound, an ectopic pregnancy is very likely. The value is above 2000 mIU/ml with vaginal ultrasound. This level is known as the discriminatory zone. In this case ultrasound and β -HCG assay are repeated. In normal pregnancy the hormone level doubles after 3 days and increases by at least 66% after 2 days. A subnormal rise favours ectopic pregnancy as the production of hormone is impaired.
8. *Laparoscopy.* It confirms the diagnosis in case of doubt. It is not done in the presence of excessive intraperitoneal haemorrhage as these cases need immediate laparotomy.
9. *Culdocentesis.* Needling of Douglas pouch to diagnose pelvic haematocoele.
10. *Computed tomography.* It confirms abdominal pregnancy if ultrasound is not conclusive.
11. *Magnetic resonance imaging.* Very accurate to diagnose abdominal pregnancy.
12. *Uterine curettage.* Examination of the products from the uterus shows decidua cells but no chorionic villi. Sometimes, the endometrium shows the Arias-Stella reaction. It is done when we suspect incomplete abortion and the criteria are: (a) if β -HCG level does not rise in the normal way, (b) serum progesterone 5 nanograms/ml or less indicating a dead ovum. Also, if the patient passes a piece of tissue via the vagina, it is examined pathologically for chorionic villi.

Treatment of Tubal Pregnancy

1. Undisturbed Ectopic

It is treated by one of the following methods:

- a. *Laparotomy.* Both tubes are inspected because bilateral tubal pregnancy may occur, also the other tube may be diseased, absent or malformed. The treatment is salpingectomy or the tube is incised, contents evacuated and tube is either closed (salpingotomy) or left open to heal by secondary intention (salpingostomy). Pregnancy in the isthmic part of the tube is treated by segmental tubal resection and anastomosis of the tubal segments. Conservative tubal surgery is better than salpingectomy as it improves future fertility.
- b. *Laparoscopy.* To avoid laparotomy the laparoscope is used to perform any of the above procedures. It can be used to inject prostaglandin (E_2 or $F_{2\alpha}$), methotrexate (50 mg), potassium chloride (20%), or hyperosmolar glucose (50%) solution into the tubal mass to destroy the conceptus (salpingocentesis).

- c. Under ultrasound guide a needle is passed to inject one of the above substances.
- d. To give systemic methotrexate. This medical treatment leads to spontaneous resorption of the ectopic pregnancy in 90–95% of cases.

With conservative management, the patient is followed up by repeated estimation of serum beta-HCG twice weekly to detect active remnants of trophoblastic tissue.

2. Subacute type

Laparotomy followed by salpingectomy of the affected tube. Salpingo-oophorectomy is done if the ovary is markedly destroyed or when it is impossible to separate it from the tube. Blood is then removed from abdominal cavity before abdominal closure. To avoid laparotomy, the laparoscope is used to perform salpingectomy.

3. Acute type

Rapid antishock measures followed by immediate laparotomy. The affected tube is clamped immediately to stop the bleeding then it is removed. Do peritoneal toilet and inspect the other tube before closure of the abdomen. Antishock measures are continued during and after operation.

4. Chronic type with pelvic haematocele

Laparotomy is performed, blood is evacuated from Douglas pouch and the affected tube is removed.

5. Abdominal Pregnancy

Once the condition is diagnosed laparotomy is performed because the fetus is malformed in 50% of cases, and there is a risk of infection or rupture and haemorrhage. The fetus, placenta and tube are removed. If the placenta is attached to important structures as the intestines or large blood vessels, it is dangerous to remove it, and so the cord is cut very short, ligated with silk, and the placenta is left to become absorbed within 1 or 2 years. However, if the placenta is attached to a removable structure, e.g. omentum it can be removed with it. If the placenta is allowed to remain methotrexate may be given to destroy the trophoblast and speed resorption of the placenta.

N.B.:

- If the woman is Rh negative and not sensitized anti-D serum is given. 100 micrograms if pregnancy is less than 12 weeks, and 300 µg if more than 12 weeks.
- Methotrexate is given in the same dose as in choriocarcinoma, or given as a single IM injection of 50 mg/m² of body surface area.

OVARIAN PREGNANCY

It is very rare (1:25,000 pregnancies). The clinical picture is the same as tubal pregnancy. At operation the criteria of ovarian pregnancy (Spiegelberg criteria) are:

1. The pregnancy sac occupies the position of the ovary;

2. It is connected to the uterus by the ovarian ligament;
3. The wall of the sac must contain ovarian tissue;
4. Both tubes must be normal.

It is treated by laparotomy and removal of the affected ovary, if the mass alone cannot be removed.

INTERSTITIAL OR CORNUAL PREGNANCY

It is pregnancy in the interstitial part of the tube which traverses the uterine wall. Rupture occurs at 10 to 14 weeks. Treated by wedge resection or hysterectomy depending upon the degree of damage to the uterus.

PREGNANCY IN A RUDIMENTARY HORN

Rupture occurs at 16 to 20 weeks or later due to the presence of myometrium. Treated by excision of the horn. At operation pregnancy in a rudimentary horn may be mistaken for pregnancy in the interstitial part of the tube. The round ligament is inserted lateral to the first and medial to the second.

CERVICAL PREGNANCY

Ovum is implanted in the cervical canal. Bleeding may be severe because the cervix is non-retractile and pain is absent. Cervix is enlarged and soft with products of conception in the cervical canal.

Treatment

Surgical

1. Suction evacuation of the products of conception and curettage. To reduce bleeding haemostatic cervical sutures are placed at 3 and 9 o'clock before the procedure, followed by insertion of a Foley catheter into the cervical canal to compress the bleeding area (30 ml catheter bulb) or a silk ligature is used to surround the cervix similar to a McDonald cerclage. Also uterine artery embolization with Gelfoam can be done prior to suction evacuation.
2. Hysterectomy if severe bleeding can not be controlled.

B) Medical

Injection of methotrexate into the gestational sac to induce fetal death or the drug is given systemically. ..

OTHER TYPES

Bilateral tubal pregnancy. Combined intra and extrauterine pregnancy (heterotopic pregnancy). Incidence of the latter varies from 1 in 3000 to 1 in 30,000 pregnancies. Incidence increases with assisted reproductive technology (ART) to be about 1 in 100 pregnancies.

THE DIFFERENTIAL DIAGNOSIS OF ABORTION AND ECTOPIC PREGNANCY

<i>Abortion</i>	<i>Ectopic Pregnancy</i>
1. Bleeding occurs before the pain	1. Bleeding occurs after the pain.
2. Blood is bright red in colour.	2. Usually dark brown (prune juice)
3. Pain is colicky, felt in the suprapubic region and accompanied with backache.	3. Pain may be dull aching, sharp stabbing or colicky and is felt in one iliac fossa.
4. If shock is present, it is related to the amount of vaginal bleeding.	4. Shock is not related to the amount of vaginal bleeding.
5. No abdominal tenderness or rigidity.	5. Abdominal tenderness and rigidity.
6. Vaginal fornices are not tender.	6. Very tender.
7. Size of uterus corresponds to the period of amenorrhoea.	7. The size of 8 weeks pregnancy.
8. No adnexal swelling.	8. Very tender adnexal swelling.
9. Microscopic examination of the products from the uterus shows decidual cells and chorionic villi.	9. Decidual cells but no chorionic villi.

A SHORT ACCOUNT ON THE DIAGNOSIS OF ECTOPIC PREGNANCY

1. *History:* There is usually a history of primary or secondary infertility. A history of recurrent attacks of lower abdominal pain, fainting and vaginal bleeding suggests disturbed ectopic. Vaginal bleeding occurs after pain while in abortion bleeding occurs before the pain.
2. *Clinical Examination:* The general, abdominal and vaginal examinations vary according to the clinical type. A characteristic local finding is severe tenderness in the vaginal fornices and on movement of the cervix.
3. *Special Investigations:* As mentioned before.

N.B.

- Recurrent ectopic pregnancy occurs in about 10% of cases.
- Conception rate after ectopic pregnancy is about 50%.
- *Arias-Stella Reaction:* The cells lining the endometrial glands are increased in size, the cytoplasm becomes foamy, vacuolated and increased in amount. The nuclei are large, hyper-

chromatic, lobulated with irregular and bizarre shapes and show numerous mitoses. There is no decidual reaction in the stroma. These changes may resemble adenocarcinoma and are most probably due to hormonal overstimulation. This reaction is seen in intra- and extrauterine pregnancy, hydatidiform mole and choriocarcinoma.

GESTATIONAL TROPHOBLASTIC TUMOURS

Arise from the trophoblast and include:

1. Hydatidiform (vesicular) mole. Also known as molar pregnancy.
2. Invasive mole (chorioadenoma destruens).
3. Choriocarcinoma.
4. Placental site trophoblastic tumour.

HYDATIDIFORM MOLE

It is a benign tumour of the chorionic villi, characterized by:

1. Marked proliferation of the trophoblast, both the syncytium and cytotrophoblast are affected.
2. Oedema or hydropic degeneration of the connective tissue stroma of the villi which leads to their distension and the formation of vesicles.
3. Avascularity of the villi: The blood vessels disappear from the villi which explains the early death of the embryo.

Incidence

It varies in different parts of the world. It is high in Asia. About 1 in 1000 pregnancies in United States and Europe and about 1 in 80 pregnancies in Taiwan. Predisposing factors include race, deficiency of protein or carotene in the diet and maternal age (<20 or >40).

Pathology

1. Uterus

It is usually larger than the period of amenorrhoea. It is distended with a large number of vesicles which are closely packed together. They vary in size, some are minute 2 mm in diameter, others are large reaching 2 cm or more. Each vesicle has a fine pedicle. The fluid content is clear and watery. The condition occurs in the early weeks of pregnancy (3rd to 5th week) leading to death and absorption of the fetus or it may remain as a rudimentary structure. In rare cases the disease starts late in pregnancy when only a part of the placenta is affected and in this case the fetus and the placenta can be recognized (partial mole). Also in binovular twins, one ovum only may be affected and so the other fetus and placenta can be recognized. The resemblance to hydatid cyst originated the term hydatidiform mole.

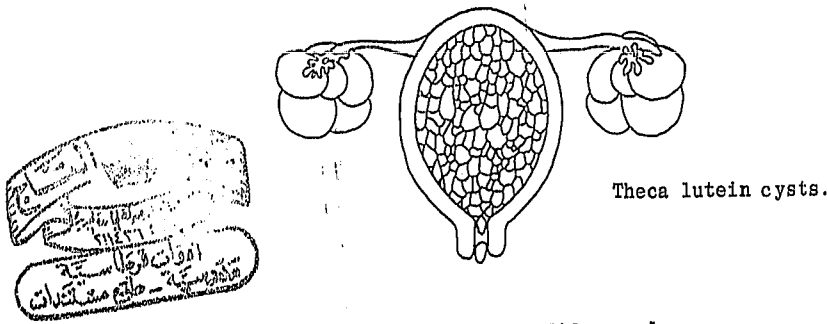


Fig. 34. Hydatidiform mole

2. The Ovaries

In about 30–60% of cases one or both ovaries contain multiple theca lutein cysts which may reach a large size (10 cm or more). They are due to stimulation of the ovaries by the excessive human chorionic gonadotrophin (HCG) produced by the proliferated trophoblast. Cysts disappear within few months (2–3), after evacuation of the mole.

Note: *Invasive Mole or Chorioadenoma Destruens:* It is a trophoblastic tumour. Hydropic chorionic villi with absent fetal blood vessels and with marked proliferation of the trophoblast are seen invading the myometrium. It is locally malignant and rarely metastasizes. It may lead to perforation of uterus.

Diagnosis

1) Symptoms

1. Symptoms of early pregnancy as amenorrhoea and morning sickness, vomiting is usually excessive (hyperemesis gravidarum — 20 to 30%).
2. Symptoms of pre-eclampsia may be present as headache, and oedema.
3. Abnormal abdominal enlargement due to distension of the uterus with vesicles.
4. Vaginal bleeding. It is the main complaint. It is due to separation of vesicles from the uterine wall. There may be a blood stained watery discharge, the watery part is from ruptured vesicles. Prune juice discharge may occur, the blood is brown because it has been retained for some time in the uterine cavity. The passage of vesicles is diagnostic. The blood may be concealed causing enlargement and tenderness of the uterus.
5. Pain, its causes are:
 - a) Rapid distension of the uterus by the mole or by concealed haemorrhage leading to dull aching abdominal pain.
 - b) A perforating mole causes localized pain and tenderness on the uterus.
 - c) Ovarian pain due to stretching of the ovarian capsule or complication in the cystic ovary as torsion.

II) Signs

A) General Examination

1. Signs of pre-eclampsia may be present (30%) denoted by hypertension, oedema and/or proteinuria.
2. Signs of anaemia may be present due to bleeding.
3. Hyperthyroidism occurs in 3–10% of cases due to chorionic thyrotrophin secreted by trophoblast also HCG has a thyroid-stimulating effect.
4. Breast signs of pregnancy.

B) Abdominal Examination

1. The uterus is often larger than the period of amenorrhoea (50%). In some cases, the mole ceases to grow and so the uterus may correspond to the period of amenorrhoea (35%) or may be smaller (15%).
2. The uterus is doughy in consistency due to absence of amniotic fluid and its distension with vesicles.
3. Absence of fetal parts and fetal heart sounds (unless there is a fetus and a mole as in case of twins and partial mole).
4. Absence of external ballottement.

C) Vaginal Examination

1. Local signs of pregnancy.
2. The presence of vesicles is a sure sign.
3. No internal ballottement.
4. One or both ovaries may be felt enlarged and cystic.

III) Investigations

1. Ultrasonography gives a characteristic picture (snowstorm appearance) and no fetus is seen.
2. Plain X-ray to the abdomen reveals absence of the fetus. Done if there are no facilities for ultrasound.
3. Very high serum level of HCG (more than 100,000 mIU/ml). The result is compared with the level for normal pregnancy at the same stage.
4. X-ray of the chest should be performed in every case of trophoblastic tumour.



Fig. 35. Ultrasound appearance of hydatidiform mole

Differential Diagnosis

1. Other causes of an oversized pregnant uterus as multiple pregnancy.
2. Causes of bleeding in early months of pregnancy as abortion.

Complications

1. Haemorrhage.
2. Perforation of the uterus, spontaneous by a perforating mole or during evacuation.
3. Uterine infection, due to the large surface area left after expulsion or evacuation of the mole.
4. Development of choriocarcinoma in about 5% of cases and invasive mole in about 10% of cases.
5. The associated pre-eclampsia may end in eclampsia.
6. Hyperthyroidism (3-10%).
7. Disseminated intravascular coagulation. Thromboplastin may be released from the trophoblast into the maternal circulation.
8. Trophoblastic embolization leading to dyspnoea and cyanosis. This occurs as a result of evacuation in 2-3% of cases.
9. Recurrent mole may occur (1-2%).

Treatment

Once the diagnosis is confirmed, the molar pregnancy is terminated to avoid complications. Blood must be ready for transfusion because severe haemorrhage may occur at any time (at least one litre of whole blood).

1. Suction Evacuation

It is the most safe and effective method of evacuation of the mole and performed irrespective of the size of the uterus. An oxytocin drip is given during operation to reduce blood loss and protect against perforation of uterus (20 units in 500 ml 5% glucose in normal saline, 30 drops/minute). After the mole is evacuated the uterus is gently curetted with a sharp curette. The material removed is sent for histopathological examination. Rh-negative women who are not sensitized should receive Rh immunoglobulin.

2. Abdominal Hysterectomy

It is preferred if the patient is above 40 years, because the incidence of choriocarcinoma is high above this age (35%) and in some cases of perforating mole.

Note: The theca lutein cysts are hormone-dependent and will disappear spontaneously after evacuation of the mole so they are not removed surgically unless complication arises as torsion or rupture.

Follow up

1. The material removed from the uterus is examined histologically to exclude malignancy.
2. The beta-subunit of serum HCG is measured by radioimmunoassay every week till the test becomes negative for 3 successive weeks then the test is repeated every month for one year. Pregnancy is allowed if the test remains negative for one year.
3. Physical and pelvic examination every 2 weeks until normal then every 3 months.
4. Pregnancy is prevented by a combined contraceptive pill or by a mechanical method (e.g. the male condom). The intrauterine device is not used because it may lead to irregular uterine bleeding which confuses the follow up.
5. Chemotherapy is started if the level of serum HCG rises (doubling in 2 weeks) or plateaus (failure to decrease over 3 weeks) or the test for the hormone becomes positive after being negative or if metastases appear.

Notes for the Postgraduate

- Normally the beta-subunit of HCG reaches the normal value 8–12 weeks after evacuation of the mole. Radioimmunoassay detects as low as 1 mIU/ml of beta-subunit.
- *Complete Hydatidiform Mole*: Fetus is absent. An empty ovum (without a nucleus-blighted) is fertilized by a haploid sperm (23X) which duplicates its chromosomes or by 2 sperms (23X and 23Y), so all chromosomes are of paternal origin. Most complete moles are 46XX (90%) and few (10%) are 46XY. It turns malignant in about 15% of cases.
- *Incomplete (Partial) Mole*: Fetus or fetal parts are present. It results from fertilization of a normal ovum (23X) by 2 sperms, so it is always triploid (69 chromosomes) and has both maternal and paternal chromosomes. It is XXY (70%) or XXX (27%) or XYY (3%). Malignant change is rare (1%) but follow up is advisable (for 6 months).
- The combined contraceptive pill is started when the beta-HCG becomes negative. Till this happens the condom can be used. If the pill is used early the beta-HCG will take a longer time to become negative as oestrogen stimulates the growth of trophoblast.
- Hormones secreted by the mole. Oestrogens, progesterone, human chorionic gonadotrophin, chorionic thyrotrophin, and human placental lactogen.

VOMITING OF PREGNANCY**I) MORNING SICKNESS OR EMESIS GRAVIDARUM****Definition**

Nausea and sometimes vomiting especially in the morning but it does not affect the general condition of the pregnant woman. It is common, occurs in about 50% of pregnant women. It usually appears at the 6th week and disappears after the 12th week.

Aetiology

Unknown. There are several theories, but none of them is completely satisfactory and will be discussed later.

Treatment

1. Reassurance of the patient.
2. Diet: The meals should be small and frequent (4-6 meals). These are easily tolerated and prevent hypoglycaemia which may be a cause of vomiting. Solid foods are preferable. Increase the carbohydrate and diminish fat in the diet. The first meal is given before leaving bed in the morning and the patient is advised to stay in bed for half an hour after this meal.
3. Drugs: Antiemetics, antihistaminics, corticosteroids and vitamin B6 (25 mg orally TDS).
4. Acupuncture may help.
5. Iron therapy is temporarily stopped.

II) HYPEREMESIS GRAVIDARUM (PERNICIOUS VOMITING OF PREGNANCY)

Definition

Excessive vomiting affecting the general condition of the pregnant woman. This is manifested by dehydration, weight loss and electrolyte imbalance.

Incidence

About 0.3% of pregnancies.

Aetiology

Unknown. There are several theories, but none of them is completely satisfactory.

1. *Allergic Theory*: The patient is allergic to the corpus luteum of pregnancy, so some patients improve when given antihistaminics and after the third month when the corpus luteum degenerates.
2. *Neurotic Theory*: The most accepted theory and it is evidenced by (a) some women start vomiting for the first time after being told that they are pregnant; (b) in very rare cases the husband may vomit when his wife is pregnant; (c) improvement of the patient by isolation and reassurance.
3. *Hormonal Theory*:
 - a) Excessive human chorionic gonadotrophin (HCG): Proved by frequency of vomiting in cases of hydatidiform mole and multiple pregnancy. It also occurs early in pregnancy when there is a high level of this hormone.
 - b) Deficiency of suprarenal cortical hormones. Some patients improve on corticosteroids.
 - c) Mild hyperthyroidism with increased free T₄ (thyroxine) concentration. HCG increases thyroid activity. The condition improves when propylthiouracil is given.

4. *Deficiency Theory*: Deficiency of carbohydrates (leading to hypoglycaemia) or vitamins B₁ and B₆ may cause vomiting. These may be effects rather than causes.

Pathology

The changes in fatal cases are similar to those found in patients dying from starvation and dehydration.

1. *The brain*: It shows congestion and petechial haemorrhages, especially in the grey matter of the third ventricle. The lesions are similar to those found in Wernicke encephalopathy and are caused by vitamin B₁ deficiency.
2. *The eyes*: Examination of the fundus may show optic neuritis, retinal haemorrhage or retinal detachment.
3. *Heart*: It is small, atrophic and there may be subendocardial or subepicardial haemorrhages (Brown atrophy).
4. *Liver*: Small with fatty infiltration and necrosis starting at the center of the lobules.
5. *Kidneys*: Show degeneration of the tubules.
6. *The peripheral nerves* may show degeneration and polyneuritis.

Biochemical Changes

1. Changes in the Blood

Blood volume is diminished due to vomiting and loss of fluids. Blood viscosity is increased. Dehydration results in a raised haemoglobin concentration, and haematocrit value. Sodium, potassium and chloride are diminished. Blood urea is increased. Acidosis due to abnormal fat metabolism secondary to lack of carbohydrates. Acidosis aggravates vomiting which increases the acidosis, and a vicious circle is produced.

2. Changes in Urine

Volume is diminished. Specific gravity is increased. Chloride is low or absent. There is proteinuria and bile salts may appear (liver damage). Ketone bodies may be present.

Diagnosis

Symptoms

The main symptom is vomiting. The condition usually starts as morning sickness which becomes gradually aggravated. As a result of vomiting there is dehydration and starvation and these will lead to oliguria, constipation, weakness and fainting. Pain and tingling sensation may occur due to peripheral neuritis.

Signs

1. Signs of dehydration as dry tongue.
2. Signs of starvation so the patient is emaciated and exhausted.
3. The pulse is rapid and weak.
4. The temperature may be raised.
5. The blood pressure is low.
6. Amount of urine is diminished.
7. In advanced cases, jaundice may appear or there is evidence of Wernicke encephalopathy in the form of drowsiness and squint. Finally there is coma and death.

Investigations

1. Complete blood count. Dehydration results in a raised haemoglobin concentration and haematocrit value.
2. Urinalysis for protein, ketone bodies, bile salts, and a sample is sent for culture to exclude urinary tract infection which may be responsible for the vomiting.
3. Serum electrolytes (glucose, sodium, potassium, chloride, and bicarbonate).
4. Renal function tests (serum creatinine, and creatinine clearance).
5. Liver function tests.
6. Thyroid function tests to exclude hyperthyroidism.
7. Sonography to confirm pregnancy, and to exclude multiple pregnancy or hydatidiform mole.
8. Examination of the fundus oculi.

Treatment

1. *Hospitalization:* The patient is isolated in a separate room at hospital and no visitors are allowed.
2. Complete rest in bed and reassurance.
3. *Diet:* Nothing by mouth until vomiting stops (usually within 24–48 hrs). The fluid and caloric requirements are given intravenously in the form of glucose and saline solutions. When vomiting is controlled feeding by mouth is gradually started by small solid meals rich in salt and carbohydrates and poor in fat.
4. *Drugs:* Sedatives and tranquilizers for neurosis. Antiemetics, antihistaminics, corticosteroids, vitamin B₁ and B₆. Also ginger can be given (a capsule containing 250 mg ginger is given 4 times daily, i.e., 1 g daily).
5. Acupuncture may help.
6. Iron therapy is temporarily stopped.
7. Psychotherapy is needed in some cases.

8. Observation:

- a) Vital signs (pulse, temperature and blood pressure) daily.
- b) Body weight daily.
- c) Urine analysis daily.
- d) Serum electrolytes daily (sodium, potassium, chloride, bicarbonate and serum creatinine).
- e) Frequency, amount and character of vomiting.
- f) Fluid chart, fluid intake and urine output daily.
- g) Examination of fundus oculi weekly.

9. Termination of pregnancy: It is indicated in the following conditions:

- a) Persistence of vomiting in spite of treatment.
- b) Urinary changes in the form of progressive oliguria and increasing proteinuria.
- c) High serum creatinine.
- d) Appearance of jaundice.
- e) The presence of fundal changes.
- f) Occurrence of Wernicke encephalopathy.

Methods of Termination.

1. If pregnancy is less than 12 weeks we do vaginal or suction evacuation.
2. If pregnancy is more than 12 weeks we do abdominal hysterotomy. Anaesthetic agents toxic to the liver as fluothane are avoided. Also operation can be done under local infiltration anaesthesia.

CAUSES OF VOMITING DURING PREGNANCY

- a) *Diseases of pregnancy:* (1) Emesis gravidarum; (2) hyperemesis gravidarum; (3) disturbed ectopic pregnancy; (4) hydatidiform mole; (5) pre-eclampsia; (6) acute polyhydramnios; (7) pyelonephritis of pregnancy; (8) acute fatty liver of pregnancy.
- b) *Medical conditions* as food poisoning.
- c) *Surgical conditions* as acute appendicitis.
- d) *Gynaecological lesions* as red degeneration in a fibroid or twisted ovarian cyst.



HYPERTENSIVE DISORDERS IN PREGNANCY

CLASSIFICATION

A) Pregnancy-induced Hypertension

Hypertension that develops as a result of pregnancy and disappears after delivery.

1. Hypertension without proteinuria or pathological oedema (gestational hypertension).
2. Pre-eclampsia.
3. Eclampsia.

B) Chronic Hypertension

It is hypertension present before pregnancy or persists more than 6 weeks postpartum.

C) Chronic Hypertension with Superimposed Pre-eclampsia and Eclampsia

It means the development of pre-eclampsia or eclampsia in a woman with chronic hypertension. Diagnosis is made when the blood pressure is elevated (30 mmHg systolic or 15 mmHg diastolic) together with oedema and/or proteinuria.

Gestational Hypertension

It is the appearance of hypertension for the first time after 20 weeks of pregnancy, during labour or first 24 hrs after delivery and in absence of proteinuria or pathological oedema. This type of hypertension disappears within 10 days after delivery.

PRE-ECLAMPSIA

Definition

It is a specific condition which occurs only in the human female during pregnancy. It is characterized by hypertension, oedema and/or proteinuria (at least 2 signs are necessary for diagnosis). If untreated it may end in eclampsia which is characterized by the occurrence of convulsions. Pre-eclampsia occurs after 20 weeks of pregnancy, usually in the last 12 weeks, however, it occurs in the first half of pregnancy in cases of hydatidiform mole, multiple pregnancy, acute polyhydramnios, hydrops fetalis (rhesus and non-rhesus) and the presence of antiphospholipid antibodies.

Incidence

About 5% of pregnancies. However, the incidence varies in different parts of the world.

Aetiology

A) Predisposing Factors

1. Maternal Factors

1. Age. The disease is more common in young women below 20 and women above 35 years.

2. Parity. Primigravidae are more affected than multigravidae (2:1). Also more common in the grand multipara.
3. Past or family history of pre-eclampsia (mother or sister).
4. Maternal diseases. Chronic hypertension, chronic renal disease, diabetes mellitus, obesity, connective tissue diseases as systemic lupus erythematosus, antiphospholipid antibody syndrome, migraine, and asthma.
5. If the woman is married to a new partner due to paternal genes.

II. Fetal Factors

Multiple pregnancy, hydatidiform mole, hydrops fetalis (immune and nonimmune), polyhydramnios, and ? male fetus.

B) The Actual Cause

This is not known. There are several theories, none of them is completely satisfactory. Pre-eclampsia is the disease of theories.

Note: Smoking reduces the incidence of pre-eclampsia by 50%, possibly due to thiocyanate which reduces blood pressure, or nicotine which reduces thromboxane A₂ production.

Pathology

The lesions of pre-eclampsia are characterized by spasm of the small arterioles all over the body, thrombosis, haemorrhage and necrosis. These lesions are liable to occur in severe cases of pre-eclampsia.

1. *Brain:* Cerebral oedema, thrombosis, haemorrhage and softening (infarction) may occur.
2. *Changes in the fundus oculi:*
 - a) Papilloedema: Oedema of the optic disc.
 - b) Beading of the blood vessels due to localized spasm affecting the retinal arterioles. Arteriolar spasm is the most common retinal change associated with pre-eclampsia.
 - c) If the kidneys become affected we get the picture of albuminuric retinitis with appearance of cotton wool patches of exudate and star shaped haemorrhages.
 - d) In rare cases there is retinal detachment due to intraocular oedema. The prognosis is good because spontaneous reattachment of the retina occurs within one week after delivery.
3. *Heart:* Degeneration of the muscle fibres with areas of subendocardial haemorrhage.
4. *Lungs:* Congestion and oedema and multiple minute thrombi.
5. *Liver:* It is enlarged, areas appear yellow due to necrosis and areas appear red due to haemorrhage. The necrosis starts at the periphery of the lobule and proceeds towards the center. There is thrombosis of the interlobular vessels. HELLP syndrome may

occur. It is characterized by haemolytic anaemia, elevated liver enzymes and low platelet count. It occurs in 10% of severe cases of pre-eclampsia.

6. *Gastrointestinal tract*: Congestion and oedema.
7. *Kidneys*: On cut section the cortex is pale and the medulla is congested. Microscopically the glomeruli are slightly enlarged, the capillary endothelial cells are swollen (endotheliosis) and a fibrinoid material is deposited between the cells and the basement membrane. There is degeneration of the tubules which may contain casts. In rare cases there is tubular or cortical necrosis. The glomerular filtration rate is reduced by 25% in mild cases and 50% in severe cases. The renal clearance of uric acid is reduced subsequent to reduced renal blood flow, also the production of uric acid is increased due to tissue damage resulting from ischaemia, so its serum level is increased. Hyperuricaemia differentiates pre-eclampsia from other causes of hypertension during pregnancy. A serum level above 5 mg/dl diagnoses pre-eclampsia and a level above 6 mg/dl suggests severe pre-eclampsia.
8. *Adrenals*: Thrombosis, haemorrhage and necrosis.
9. *Uterus*: This may show multiple and extensive placental infarcts, retroplacental haematoma, with thrombosis, haemorrhage and necrosis in the muscle layer. There is no specific placental lesion typical of pre-eclampsia.
10. *Other changes* include reduced plasma volume, haemoconcentration, reduced plasma proteins, diminished production of progesterone and oestriol. Some patients develop disseminated intravascular coagulation (DIC) due to damage of the capillary endothelial cells and release of thromboplastin from the degenerated placenta. Also antithrombin III may be decreased secondary to severe proteinuria.

Clinical Picture

In case of pre-eclampsia, the signs appear before the symptoms. Symptoms appear in severe cases.

A) Signs

1. *Hypertension*: It is usually the first sign. It is due to vasospasm. Hypertension is diagnosed during pregnancy when the systolic blood pressure is 140 mmHg or above, or if the systolic pressure is elevated 30 mmHg or more above the previous level. Hypertension is also diagnosed when the diastolic pressure is 90 mmHg or above, or if it is elevated 15 mmHg or more above the previous level. The blood pressure should be recorded on at least 2 occasions 6 hours or more apart, with the patient resting in bed.
2. *Oedema*:
It may be due to reduction in the level of serum albumin or increased capillary permeability due to damage of capillary endothelial cells. There are 2 types:

- a) **Manifest or clinical oedema:** It is oedema of the subcutaneous tissues. Oedema of the feet and ankle region may be physiological and is due to pressure of the pregnant uterus on pelvic veins. However, oedema, spreading along the leg or affecting the vulva, lower abdominal wall, face or hands is never physiological.
- b) **Occult oedema:** It is oedema of the internal organs. It is suspected if there is abnormal increase in body weight. Weight gain more than 1 kg/week or 3 kg/month is abnormal. Sudden and excessive weight gain may be the first sign of pre-eclampsia.
3. **Proteinuria:** It is a late sign. It is always preceded by hypertension. Proteinuria means the presence of 300 mg or more of protein in 24-hour collection of urine. It is due to glomerular damage allowing a leak of proteins. The proteinuria is essentially albuminuria.

B) Symptoms

The following symptoms may occur in severe cases:

1. Headache due to cerebral oedema and hypertension. Often frontal but may be occipital.
2. Eye symptoms as blurring of vision and diplopia.
3. Nausea and vomiting due to oedema of the brain, oedema of the gastric mucosa or affection of the liver.
4. Epigastric pain due to enlargement of the liver and stretching of its capsule.
5. Oliguria and anuria due to kidney pathology.

N.B.: The diagnosis of pre-eclampsia is easy if the above mentioned signs and symptoms appear in the second half of pregnancy in a patient who was normal from the start of pregnancy. However, if the case is seen for the first time late in pregnancy the condition must be differentiated from other causes of hypertension, oedema and proteinuria during pregnancy.

Types of Pre-eclampsia

Pre-eclampsia may be mild or severe. In severe cases one or more of the following is present:

1. A systolic blood pressure 160 mmHg or above or a diastolic pressure 110 or above.
2. Massive proteinuria: 5 gms or more in 24 hours or a 3 plus qualitative test.
3. Oliguria or anuria.
4. HELLP syndrome.
5. The presence of symptoms as headache, vomiting, etc.
6. Pulmonary oedema.

Note. Oliguria means urine output less than 400 ml/day, and anuria when output is less than 100 ml/day

Imminent or Impending Eclampsia

It is a severe form of pre-eclampsia in which symptoms as headache and vomiting are accompanied by a marked increase in the physical signs. This condition lasts for a few hours followed by convulsions of eclampsia.

Prognosis of Pre-eclampsia

A) Maternal Complications

The following complications may occur: (1) Eclampsia (1–2% of cases); (2) cerebral haemorrhage; (3) heart failure and pulmonary oedema; (4) liver failure; (5) renal failure due to tubular or cortical necrosis; (6) abruptio placentae (in 10% of severe cases); (7) retinal detachment; (8) haemolytic anaemia; (9) disseminated intravascular coagulation (DIC); (10) liability of recurrence of pre-eclampsia in subsequent pregnancies (15%).

Maternal mortality is 0–5%.

B) Fetal Complications

1. Intrauterine growth restriction.
2. Intrauterine fetal death due to multiple placental infarctions and premature separation of the placenta by retroplacental bleeding (abruptio placentae). The fetus is in danger if the systolic pressure exceeds 160 or the diastolic exceeds 110 because at this level placental abruption is liable to occur.
3. Prematurity and its complications as respiratory distress syndrome, haemorrhage and infection in the newborn.

Manifestations of pre-eclampsia improve when the fetus dies inside the uterus.

Treatment

A) Prophylactic Treatment

1. Routine antenatal care to allow early detection of the disease to avoid maternal and fetal complications.
2. Women who are liable to develop pre-eclampsia as diabetic and hypertensive patients are seen more frequent than normal.
3. All women are asked to report immediately any of the symptoms of pre-eclampsia, such as headache, visual disturbances and oedema of the face or hands.
4. Patients with severe hypertension or renal disease are advised against becoming pregnant.
5. A small dose of aspirin 75 mg tablet is given daily from the 12th week to the woman with a past history of pre-eclampsia, or whose mother or sister had the disease. It is stopped at 38 weeks as it delays onset of labour. However, the value of this baby

aspirin is not definitely proven. Low-dose aspirin inhibits production of thromboxane A₂ from platelets with minimal effects on prostacyclin produced by the endothelium of blood vessels. Thromboxane A₂ causes vasoconstriction and aggregation of platelets favouring thrombosis. Prostacyclin has the opposite effect.

6. Calcium supplementation as 2 g orally daily from week 15 to 38th week leads to reduction in systolic and diastolic blood pressure and pre-eclampsia. Calcium reduces blood pressure by decreasing the sensitivity to angiotensin II, or by reducing the production of parathyroid hormone thus reducing the intracellular calcium ion. This leads to relaxation of smooth muscle fibres and vasodilatation. The benefit is also controversial.

B) Treatment of Pre-eclampsia

1. *Hospitalization* : All cases of pre-eclampsia are admitted to hospital as the progression of the disease is unpredictable.
2. *Complete rest in bed* for most of the day. Rest in bed lowers the blood pressure, stimulates diuresis, relieves oedema, increases renal and placental blood flow. The lateral position is preferred to the dorsal one to avoid compression of the inferior vena cava and aorta by the pregnant uterus.
3. *Normal diet*, rich in protein (2 g/kg body weight) is given. Salt is neither restricted nor encouraged so foods rich in salt as sardines, tuna and canned foods are avoided.
4. *Antihypertensives* are given in severe cases if systolic pressure is 160 mmHg or above or diastolic 110 mmHg or above. In severe cases, these drugs are given by intravenous drip. The commonest drugs used are methyldopa (Aldomet), hydralazine (Apresoline), labetalol, and nifedipine.
5. Some obstetricians give magnesium sulphate in severe cases to prevent occurrence of eclamptic fits.
6. *Observation of the case*:
 - Blood pressure is recorded every 6 hours and more frequently in severe cases.
 - Fetal heart sounds are heard every 6 hours.
 - Urine is examined daily for amount and protein.
 - Oedema is assessed daily.
 - Body weight is recorded daily.
 - Complete blood count to exclude anaemia and thrombocytopenia.
 - 24-hour urine collection for total proteins done weekly.
 - Kidney function tests (serum creatinine, creatinine clearance and uric acid) are done once weekly and twice weekly in severe cases.
 - Liver function tests and serum albumin are determined once weekly.

- Examination of fundus oculi once weekly.
- Study of coagulation factors in severe cases (these fall with DIC).
- Tests to assess fetal growth and well-being:
 - Fetal growth is determined by clinical examination and ultrasound every 1-2 weeks.
 - Fetal well-being is assessed by daily fetal movements count, and the nonstress test which is done twice weekly or daily according to the severity of the condition. If the nonstress test is nonreactive, the contraction stress test or fetal biophysical profile is performed. Also Doppler ultrasound to study the flow in the uterine and umbilical arteries gives an idea about fetal well-being.

7. Termination of Pregnancy

Pregnancy is allowed to continue as long as the maternal and fetal conditions are good.

The following are indications to terminate pregnancy:

Fetal: (a) All cases reaching 38 weeks of pregnancy (even mild cases as placental infarction may be progressive); (b) intrauterine growth restriction; (c) fetal asphyxia detected by fetal heart rate monitoring.

Maternal: (a) Rising serum creatinine; (b) HELLP syndrome; (c) severe cases of pre-eclampsia if there is no marked improvement within 24 hours of treatment.

Note: If we need to terminate pregnancy before 34 weeks, amniocentesis is done to determine the L/S ratio for lung maturity.

Methods of Termination of Pregnancy

Artificial rupture of membranes and oxytocin drip or prostaglandin vaginal tablet or gel or caesarean section. The method depends on the condition of the mother (severity of pre-eclampsia) and the cervix.

C) Management during Labour

1. Proper sedation of the mother and repeated estimation of her blood pressure.
2. Continuous monitoring of fetal heart sounds to detect fetal distress.
3. Forceps may be needed to shorten the second stage (if prolonged more than 20 minutes).
4. Ergometrine is contraindicated in the third stage as it may raise the blood pressure leading to eclampsia. Oxytocin is given to prevent postpartum haemorrhage.

D) Management after Delivery

A sedative as diazepam (Valium) is given for 24-48 hours to avoid rise of blood pressure and postpartum eclampsia. Urine is observed for amount and protein. Treatment and observation are continued until the blood pressure returns to normal.

Note: Diuretics are not used in pre-eclampsia as they decrease the renal and uteroplacental blood flow, and increase the already present haemoconcentration favouring thrombosis.

ECLAMPSIA

Definition

It is pre-eclampsia complicated by fits.

Incidence

It is 1 in 500 to 1 in 1,500 pregnancies.

Pathology

The same as found in severe cases of pre-eclampsia.

Clinical Picture

It is the clinical picture of pre-eclampsia plus fits. The fit may follow stimuli of any kind; visual, auditory or touch. The fit occurs in 4 stages without interval in between.

1. Premonitory Stage

The patient loses consciousness, the pupils are dilated, the eyes move from side to side. Muscular twitches occur in the tongue, face and hands. This stage lasts about half a minute.

2. Tonic stage

All the voluntary muscles of the body undergo tonic contraction. The respiratory muscles are affected causing cyanosis, the muscles of swallowing are affected leading to accumulation of saliva in the mouth. The patient may acquire certain positions as opisthotonus due to spasm of the back muscles. This stage lasts about half a minute.

3. Clonic Stage

The voluntary muscles of the body undergo irregular, intermittent contractions and relaxation. The tongue may be bitten. The patient may pass faeces or urine involuntarily. It lasts about one minute.

4. Stage of Coma

This may last for minutes, hours or days. A new fit may start while the patient is still in coma or after recovery from coma. The temperature rises during a fit because of increased muscular activity.

Types of Eclampsia

1. *Antepartum eclampsia*: The fits start during pregnancy before the onset of labour (50%).

2. *Intrapartum eclampsia*: The fits start for the first time during labour (25%).
3. *Postpartum eclampsia*: The fits start after delivery, usually within 12 hours and very rarely after 48 hours (25%). However, eclampsia may occur up to 10 days postpartum.

Differential Diagnosis

- (1) Epilepsy; (2) Hysteria; (3) Uraemia; (4) Strychnine poisoning; (5) Brain tumour; (6) Intracranial haemorrhage; (7) Cerebral vein thrombosis; (8) Meningitis; (9) Encephalitis; (10) Hypertensive encephalopathy; (11) Water intoxication.

Causes of Convulsions in Eclampsia

Unknown, however, it may be due to cerebral oedema, cerebral vasoconstriction leading to ischaemia and cerebral hypoxia, platelet thrombi or retention of sodium ions causing cerebral irritability. It is not due to hypertensive encephalopathy because it may occur in women with normal blood pressure (fulminating eclampsia) and this happens in 20% of cases.

Prognosis

A) Fetal Complications

The perinatal mortality is about 10–15% and it is due to:

1. Fetal hypoxia due to multiple placental infarctions, premature separation of the placenta by accidental haemorrhage and maternal hypoxia during convulsions.
2. Prematurity and its complications as respiratory distress syndrome, haemorrhage and infection in the newborn.

B) Maternal Complications

1. Maternal mortality in 0–10% of cases.
2. Liability to recurrence of eclampsia in subsequent pregnancies (less than 5%).

Causes of Maternal Mortality

1. Cerebral haemorrhage; due to sudden rise of blood pressure during a fit. Commonest cause (70%).
2. Heart failure; due to exhaustion from repeated fits.
3. Asphyxia; due to prolonged tonic stage, falling of the tongue backwards during coma, accumulation of saliva in the pharynx or inhalation of vomitus.
4. Aspiration pneumonia.
5. Liver failure.
6. Renal failure.
7. Abruptio placentae (accidental haemorrhage).

Signs of Bad Prognosis

Eclampsia is considered severe with a bad prognosis if two or more of the following signs are present (Eden criteria).

1. Number of fits more than 10.
2. Duration of coma more than 6 hours.
3. Pulse rate above 120 beats per minute.
4. Temperature above 39° C (probably due to cerebral haemorrhage).
5. Systolic blood pressure above 200 mmHg.
6. Respiratory rate over 40/minute.
7. Absence of oedema (dry eclampsia) indicating severe vascular spasm. Oedema is absent in 30–40% of cases.
8. Excessive proteinuria. Proteinuria is absent in 20% of cases.
9. Anuria.
10. Presence of jaundice indicating liver failure.
11. Type of eclampsia; antepartum eclampsia is the most fatal because it may pass into postpartum eclampsia.

Treatment

A) General Treatment

1. The patient should be treated at hospital. She is isolated in a special semi-dark room and all noises are prevented.
2. A well trained nurse must be present beside the patient all the time.
3. The patient lies on her side and if on her back the head is tilted to one side to prevent inhalation of vomitus or saliva.
4. A catheter is fixed in the bladder for collection of urine.
5. During a fit, we put a mouth gag to avoid biting the tongue. It is removed after the fit because it interferes with swallowing.
6. Saliva and mucus should be removed from the mouth and nose.
7. Oxygen is given after the fit till cyanosis disappears.
8. During the stage of coma a tongue forceps is applied to prevent obstruction of the airway.
9. Observation: The pulse, temperature, blood pressure, respiratory rate, patellar reflex, and FHS are recorded every hour on a special chart. Urine is examined for amount and protein hourly. Also record the time and number of fits, duration of coma and the presence of uterine contractions.
10. CT scan or MRI is not done routinely, but it is indicated if the patient has local neurological signs to exclude cerebral haemorrhage.

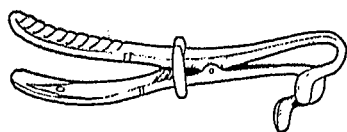


Fig. 36. Mouth gag

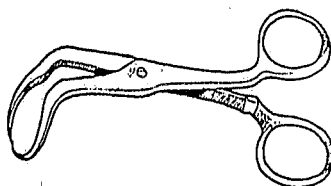


Fig. 37. Tongue forceps

B) Medical Treatment

1. Anticonvulsant drugs using magnesium sulphate solution, diazepam (Valium) or phenytoin:
 - a. Magnesium sulphate solution. It is given intravenously. The intramuscular route is easily used but painful and the serum level is not constant. The loading dose is 6 grams given slowly intravenously over 20 minutes to avoid cardiac and respiratory depression and even arrest. The infusion is then continued at a rate of 1–2 g per hour. If there is no response increase the rate up to 4 g per hour. The aim is to keep the serum level at 4–6 mg/dl ($\text{mg/dl} = \text{mEq/L} = \text{mmol/L}$). The total amount should not exceed 24 grams in 24 hours. The infusion is given until fits are controlled, and continued for 24–48 hours after delivery to prevent recurrence of fits. The mechanism of the anticonvulsant action of magnesium sulphate is unknown. The toxicity is cardiac and respiratory depression and even arrest due to muscular paralysis. The antidote is calcium which is given intravenously as 10 ml of 10% calcium gluconate solution (1 g). Magnesium sulphate is stopped and the antidote is given if (a) knee jerk (patellar reflex) becomes sluggish or absent, (b) respiratory rate becomes less than 16 per minute, and (c) amount of urine is less than 30 ml per hour. Magnesium sulphate is the best anticonvulsant.
 - b. Diazepam: 20–40 mg are given slowly intravenously (one minute for each 5 mg to avoid cardiac and respiratory arrest) followed by an intravenous drip of 40 mg/500 ml glucose 5% given at a rate of 10 mg per hour (30 drops/minute).
 - c. Phenytoin. It is given by intravenous infusion; 15 mg/kg/day up to a maximum of 1250 mg daily.
2. Anti-hypertensive drug as hydralazine given I.V.
3. Intravenous glucose: 500 ml of 5% solution given I.V. every 4 hours. This supplies calories and fluid for the patient in coma, stimulates diuresis, relieves cerebral oedema and supports the liver.
4. Digitalization: If there is heart failure.

C) Obstetric Treatment

1. In many cases labour starts spontaneously. In this case, the membranes are ruptured when the cervix is half dilated to enhance delivery. When the cervix becomes fully dilated the fetus is delivered immediately by forceps or breech extraction.
2. If the fits are controlled and the patient is stable with the blood pressure under control and the fetus is alive, we terminate pregnancy because the fits may recur. Termination is by caesarean section or rupture of membranes and oxytocin drip if the cervix is favourable. If the fetus is dead, nothing is done and we wait for its spontaneous expulsion.

D) Management after Delivery

Anticonvulsant therapy is continued for 24–48 hours after delivery to prevent recurrence of eclampsia. Postpartum eclampsia very rarely occurs 48 hours after delivery. Observation and treatment are continued until the blood pressure returns to normal. Urine is observed for amount and protein.

Notes

- The mechanism of anticonvulsant action of magnesium sulphate is unknown. However, it may interfere with the release of acetylcholine at the motor end plate, i.e., the neuromuscular junction (curare-like action) or it may displace intracellular calcium which is necessary for transmission of the nerve impulse, or it may dilate the cerebral blood vessels preventing brain ischaemia and hypoxia.
- The patellar reflex is lost (first sign of toxicity) if the serum level of magnesium sulphate is 8–12 mg/dl, respiratory depression at about 15 mg/dl, and cardiac arrest at about 30 mg/dl.
- Diuretics are not used in pre-eclampsia and eclampsia as they increase the haemoconcentration which is already present thus favouring thrombosis. Only used if there is pulmonary oedema.
- Sedatives are not recommended in pre-eclampsia as they may lead to a nonreactive nonstress test giving a wrong impression of fetal distress.
- Intercurrent eclampsia. In some cases of eclampsia the fits are controlled and pregnancy continues. If fits recur in the same pregnancy, it is called intercurrent eclampsia.
- Atypical eclampsia. It is eclampsia starting before 20 weeks of pregnancy or after 48 hours from delivery.
- All pregnant women with convulsions should be considered eclamptic until proved otherwise.

ESSENTIAL HYPERTENSION WITH PREGNANCY

Effect of Hypertension on Pregnancy

1. *Fetal*: Intrauterine growth restriction or intrauterine fetal death may occur due to placental insufficiency.
2. *Maternal*: There is increased incidence of abortion, preterm labour, pre-eclampsia and abruptio placentae.

Effect of Pregnancy on Hypertension

1. Hypertension may appear for the first time during pregnancy.

2. The blood pressure may drop in the second trimester but it is raised again in the third trimester. This drop is due to vasodilatation and decrease in the peripheral resistance which may be caused by prostacyclin (prostaglandin), and hormones of pregnancy; oestrogen and progesterone. Also the placenta acts as an arterio-venous shunt and decreases the peripheral resistance.

Diagnosis

See the table page (88).

Treatment

Women with severe hypertension are advised not to become pregnant. Treatment during pregnancy includes rest both physical and mental, regulation of diet, sedatives and antihypertensives. Therapeutic abortion is indicated for severe cases not responding to treatment. During pregnancy the fetal growth and well-being are assessed. Pregnancy is terminated when there is evidence of placental insufficiency to avoid intrauterine death and when the blood pressure becomes uncontrollable. Otherwise pregnancy can be continued to term.

Note: Placental insufficiency is manifested by retarded fetal growth or fetal distress due to asphyxia.

Causes of Hypertension during Pregnancy

1. Pregnancy-induced hypertension.
2. Primary or essential hypertension.
3. Hypertension secondary to different causes as:
 - a) Chronic renal disease.
 - b) Adrenal disease; pheochromocytoma, Cushing syndrome and hyperaldosteronism.
 - c) Endocrine disorders as thyrotoxicosis.
 - d) Coarctation of aorta.

CHRONIC RENAL DISEASE WITH PREGNANCY

It includes chronic pyelonephritis, chronic glomerulonephritis, interstitial nephritis (diabetes and gout), polycystic kidney, tuberculosis and renal artery stenosis.

Effect of Chronic Renal Disease on Pregnancy

Same as hypertension.

Effect of Pregnancy on Chronic Renal Disease

In general pregnancy leads to deterioration of kidney function in chronic renal diseases.

Diagnosis

See the table page (88).

Treatment

Some recommend avoidance of pregnancy if renal function is impaired by 50%. In fact the chance of successful pregnancy is reduced if serum creatinine level is equal to or more than 2 mg/dl or creatinine clearance is less than 50 ml plasma/minute. During pregnancy the renal function is assessed by protein excretion, serum creatinine and creatinine clearance together with assessment of fetal growth and well-being. Pregnancy is terminated if renal function deteriorates significantly or there is evidence of placental insufficiency to avoid intrauterine death.

Causes of Proteinuria during Pregnancy

1. Contamination of urine by vaginal discharge (commonest cause). Excluded by taking a mid-stream urine (MSU) or catheter specimen. Sometimes called false proteinuria.
2. Pre-eclampsia and eclampsia.
3. Diseases of urinary tract as cystitis and pyelonephritis.
4. Essential hypertension. Proteinuria is absent and if present it is slight and intermittent.
5. Hyperemesis gravidarum due to degeneration of kidney tubules.
6. Congestive heart failure due to hypoxia of kidneys.
7. Severe anaemia due to hypoxia of kidneys.
8. Specific fevers as typhoid.
9. Orthostatic proteinuria. Protein is present at the end of the day but is absent in the morning due to pressure of the lumbar spine on left renal vein during standing. Rare and diagnosed after excluding all other causes.

Note: Protein is detected by the use of protein strips as Albustix.

Causes of Oedema of Lower Limbs during Pregnancy

1. Physiologic oedema, due to pressure of pregnant uterus on pelvic veins. It affects only the feet and ankle region and present in about 40% of pregnant women.
2. Pre-eclampsia and eclampsia.
3. General causes of oedema as heart failure, renal and nutritional oedema.
4. Local causes as infection and thrombosis.
5. Angioneurotic oedema.

DIFFERENTIAL DIAGNOSIS OF PRE-ECLAMPSIA, CHRONIC RENAL DISEASE AND ESSENTIAL HYPERTENSION

<i>Differences</i>	<i>Pre-eclampsia</i>	<i>Chronic Renal Disease</i>	<i>Essential Hypertension</i>
<i>Frequency</i>	Common (70%)	Rare (5%)	Frequent (25%)
<i>Past History</i>	Nil or positive for pre-eclampsia	Repeated attacks of tonsillitis, acute nephritis or repeated attacks of urinary tract infection	Hypertension before pregnancy.
<i>Family History</i>	Negative or positive	Negative	Positive for hypertension
<i>Time of Onset</i>	Second half of pregnancy except in hydatidiform mole, multiple pregnancy, acute polyhydramnios and hydrops fetalis.	Proteinuria is present from the start of pregnancy.	Hypertension from the start.
<i>Blood Pressure</i>	Usually moderate rise. Never exceeds 200 mmHg without occurrence of fits.	May be normal or slightly elevated (150/90).	Usually markedly elevated.
<i>Clinical Oedema</i>	Absent, slight, or severe.	Usually slight due to polyuria.	Absent unless there is heart failure.
<i>Urine:</i>			
– <i>Proteinuria</i>	Absent, slight or massive	Slight due to polyuria.	Absent and if present it is slight and intermittent.
– <i>Amount</i>	Normal or may be diminished	Increased (polyuria).	Normal
– <i>Specific Gravity</i>	Normal or high due to oliguria.	Low and fixed around 1010.	Normal
– <i>Casts</i>	Absent or present	Many casts	Absent
<i>Kidney Functions</i>	Normal or impaired if there is kidney damage.	Always impaired	Normal, because by the time nephrosclerosis occurs the patient is beyond the childbearing age.
<i>Cardiac Enlargement</i>	Absent	Rare	Present in long standing cases.
<i>Fundus Oculi</i>	Characteristic picture	Albuminuric retinitis	Sclerosis and haemorrhage may be seen.
<i>Follow Up</i>	– Hypertension disappears within 6 weeks after delivery. – Albuminuria disappears within 6 weeks after delivery.	Condition persists	Condition persists

ANTIHYPERTENSIVE DRUGS

Two drugs are commonly used for treatment of hypertensive disorders of pregnancy, methyldopa and hydralazine. Beta-blockers and calcium channel blockers are also used.

1. *Methyldopa* (Aldomet): It inhibits the release of noradrenaline by acting on the vasopressor centers in the brain stem (central action). This leads to vasodilatation and decrease in peripheral resistance. Effect appears after 2–3 days. Dose 250 or 500 mg tablet 2–3 times daily. Dose is increased every 2 or 3 days. Maximum dose is 3 g daily.
2. *Hydralazine* (Apresoline): Causes arteriolar dilatation and decrease in peripheral resistance, increases cardiac output and renal blood flow. The oral dose is 25–50 mg every 6 hours. The drug is rarely used alone as it causes tachycardia. Better results are obtained when combined with methyldopa or beta-blocker as these will prevent the tachycardia. Hydralazine is given intravenously for rapid control of high blood pressure in severe pre-eclampsia and eclampsia.
3. *Labetalol*: It is alpha and beta blocker, 200 mg orally three times daily and can be increased. Also given IV in acute cases. Beta blockers may lead to reduced placental blood flow, intrauterine growth restriction, preterm labour, fetal bradycardia, respiratory depression and hypoglycaemia in the newborn.
4. *Nifedipine*: A calcium channel blocker taken sublingually or orally as 10–20 mg three times daily. It is a potent vasodilator, and may cause severe hypotension.

Note for the postgraduate:

Intravenous Apresoline is given by 2 methods:

- a) Intermittent method: Give 5 mg I.V. as a bolus slowly then give 5–10 mg every 15–20 minutes till the diastolic BP is 90–100 mmHg. Keep this level by repeating the injections as necessary. Marked drop in the BP reduces the uteroplacental blood flow and causes fetal asphyxia.
- b) Continuous infusion: 100 mg in a litre of 5% glucose solution given I.V. at a rate of 2 mg/hour (5 drops/minute). The rate is increased or decreased every 15–20 minutes to reach a diastolic pressure of 90–100 mmHg which is maintained.

SUPINE HYPOTENSIVE SYNDROME

It is characterized by hypotension and fainting which may occur when the pregnant woman lies on her back in the late weeks of pregnancy. The gravid uterus compresses the inferior vena cava leading to decreased venous return and cardiac output and fall in blood pressure. This occurs in about 20% of pregnant women in whom the vertebral collateral vessels cannot compensate. Also the large pregnant uterus will compress the aorta and reduce the uteroplacental blood flow.

MEASUREMENT OF BLOOD PRESSURE DURING PREGNANCY

During pregnancy, there is a hyperkinetic circulation and so the pulse sound may not disappear except at very low or even zero blood pressure. For this reason the diastolic blood pressure is indicated when the pulse sound becomes muffled (phase 4 Korotkoff sound).

ORGANIC HEART DISEASE WITH PREGNANCY

INCIDENCE

1–3% of pregnancies. Most of the cases are rheumatic (85%) and mitral stenosis is the most common lesion.

EFFECT OF PREGNANCY ON HEART DISEASE

1. Heart failure: This may occur:
 - a) During pregnancy at any stage but the incidence increases as pregnancy advances. The cardiac output reaches its maximum at 20 weeks and the high level is maintained till delivery.
 - b) During labour; due to straining in the second stage or after delivery of the placenta due to passage of placental blood into the general circulation.
2. Liability to atrial fibrillation.
3. Reactivation of the rheumatic condition may occur.
4. Bacterial endocarditis which is predisposed to by infection especially in the puerperium.

EFFECT OF HEART DISEASE ON PREGNANCY

1. Abortion
2. Intrauterine growth restriction (small-for-dates).
3. Intrauterine fetal death.
4. Preterm labour.
5. Polyhydramnios.

1–4 due to effect of hypoxia

GRADES OF HEART DISEASE (CLASSIFICATION OF NEW YORK HEART ASSOCIATION)

Patients with organic heart disease are classified into 4 grades:

- Grade (1): The patient has no symptoms and there is no limitation of activity.
- Grade (2): Symptoms in the form of dyspnoea, palpitation, or anginal pain on ordinary physical activity.
- Grade (3): Symptoms on less than ordinary activity, but the patient is comfortable at rest.
- Grade (4): Dyspnoea at rest with evidence of congestive heart failure.

Generally speaking, pregnancy makes the patient deteriorate one grade, e.g. grade 2 before pregnancy becomes grade 3 in pregnancy. However, the type of organic lesion is more important for prognosis than the functional grading because a patient with tight mitral stenosis in grade I may develop acute heart failure suddenly.

MANAGEMENT

I) During Pregnancy

Grade 1 and 2 (compensated heart)

1. They are treated as outpatients, but they are admitted to hospital at 38 weeks for rest in bed.
2. They are seen every two weeks until 28th week and then weekly. The case is seen by both the obstetrician and the cardiologist.
3. More periods of rest, 2 hours rest in bed in the afternoon and 10 hours by night.
4. Avoid abnormal gain in weight and foods rich in salt.
5. Guard against heart failure by treatment of anaemia, any infection and avoid emotional upsets and smoking.

Grade 3 and 4 (heart failure)

Termination of pregnancy if the patient is seen before 12 weeks, after that time she must be admitted to hospital throughout pregnancy. In hospital she is given the treatment of heart failure:

1. Rest in bed in the semi-sitting position (to reduce heart rate and dyspnoea).
2. Diet: salt free and diminish fluids (to decrease blood volume).
3. Diuretics.
4. Digitalis.

Indications of Termination of Pregnancy

1. Grade 3 and 4 seen in early pregnancy.
2. History of heart failure before pregnancy.
3. History of heart failure in previous pregnancy.

Termination is done vaginally and therefore must be performed before the 12th week. After that time termination is not advised because abdominal hysterotomy is more risky than allowing pregnancy to continue. Before termination we must improve the general condition of the patient by rest, and by medical treatment. In selected cases, mitral valvotomy for severe mitral stenosis can be done alternative to termination of pregnancy. However, the operation is better done in absence of pregnancy.

II) During Labour

First Stage

1. Rest in bed in the semi-setting position to aid respiration.
2. Oxygen inhalation if there is dyspnoea or cyanosis.

3. Analgesic as pethidine is given. Tachycardia resulting from labour pains causes heart failure.
4. Start antibiotic therapy. A combination of ampicillin and gentamycin can be given and repeated after 8 hours.
5. Observation of the mother and fetus.
6. Heart failure is expected to occur when the pulse rate rises above 110 per minute or respiratory rate above 24 (between contractions). In this case active management is given in form of rapid digitalization, strong diuretic, oxygen inhalation and sedation.

Second Stage

1. Patient in the semi-recumbent position.
2. Oxygen inhalation if there is dyspnoea or cyanosis.
3. An analgesic as nitrous oxide and oxygen inhalation is given.
4. Straining is avoided, because the rise of blood pressure may lead to heart failure.
5. If the second stage is not progressing rapidly (more than 20 minutes) the forceps or vacuum extractor is applied.

Third Stage

Ergometrine is avoided unless there is severe bleeding. Ergometrine raises blood pressure and may cause heart failure. However, oxytocin is given in the third stage to all patients – unless there is heart failure – to avoid postpartum haemorrhage.

The patient should be kept under observation for at least two hours after delivery.

III) During the Puerperium

1. Rest in bed for at least three weeks after delivery and more if there is heart failure.
2. Sedatives are given in the first few days of puerperium to reduce tachycardia.
3. Breast feeding is allowed, unless there is failure.
4. Antibiotics are given to guard against bacterial endocarditis. Antibiotics are started during labour and continued after delivery.

IV) Follow-up

If a patient had developed heart failure she is not allowed to become pregnant again.

N.B.:

- If caesarean section is indicated in the presence of heart failure it is done under local infiltration anaesthesia.
- Ampicillin 2 g IM or IV plus gentamycin 1.5 mg/kg IM or IV (not exceeding 80 mg) are given when labour starts and repeated after 8 hours. In case of penicillin allergy we give vancomycin instead of ampicillin as 1 g given slowly IV over 1 hour.

Notes for the postgraduate:**1. Mitral Valve Prolapse**

There is abnormal prolapse of one or both mitral leaflets into the left atrium during systole. The leaflets are excessively large and show myxomatous degeneration. It occurs in about 12% of women in the childbearing age. Most cases are asymptomatic, others experience palpitations, dyspnoea, chest pain, and syncope. Some cardiologists do not recommend prophylactic antibiotics during labour. Others give antibiotics only if the lesion is associated with mitral regurgitation, while others give antibiotics for every case as mitral regurgitation is not diagnosed easily during pregnancy.

2. Cardiomyopathy of Pregnancy

Peripartum cardiomyopathy occurs in the third trimester of pregnancy or the first 6 months after delivery in a healthy woman without previous cardiac disease. The condition is liable to recur in a subsequent pregnancy.

Incidence. One in 1500 to 1 in 4000 pregnancies.

Predisposing factors. Multiple pregnancy, pre-eclampsia, and black races.

Aetiology. Unknown, however, it may be due to viral infection, autoimmune disorders, genetic predisposition, or relaxin hormone produced during pregnancy may lead to excessive relaxation of cardiac muscle.

Pathology. There is dilatation and failure of one or both ventricles (dilated cardiomyopathy).

Other causes of heart failure should be excluded.

Treatment. Rest, digitalis, diuretics, salt free diet, and after-load-reducing agent as hydralazine which is the drug of choice in pregnancy. There is increased risk of thromboembolism, and so heparin is given in full dose if echocardiography shows a thrombus in the ventricle, or if the ventricle is excessively dilated.

Prognosis. Mortality rate is high 25–50%. Women who continue to have heart failure or cardiomegaly for more than 6 months should avoid another pregnancy as the condition is liable to recur, and the mortality rate in this case will be very high reaching 80% or more.

3. Coarctation of The Aorta

The typical finding is hypertension in both arms and normal or low blood pressure in the lower limbs. However, coarctation is often located at the level of the left subclavian artery, and so there is isolated hypertension in the right arm only. Vaginal delivery is allowed and caesarean section is done if there is an obstetric indication. Prophylactic antibiotic therapy should be given when labour starts.

Note. After-load-reducing agent is that one which causes dilatation of arterioles leading to a decrease in the peripheral resistance.

ANAEMIAS WITH PREGNANCY

Anaemia means the amount of functioning haemoglobin is reduced below normal. In female the Hb is 12–16 g/100 ml (male 14–18 g%). Anaemia is diagnosed if Hb is below 12 g% in the non-pregnant and below 10 g% in the pregnant woman.

CAUSES OF ANAEMIA

1. Decreased production of red cells (haemopoietic).
2. Increased destruction of red cells (haemolytic).
3. Blood loss (haemorrhagic).

Any type of anaemia can occur with pregnancy. However, the commonest type is iron deficiency anaemia.

1. PHYSIOLOGICAL ANAEMIA OF PREGNANCY

Normally the plasma volume, the number of red cells and the amount of haemoglobin increase during pregnancy. The increase is more in the plasma volume leading to haemodilution and physiological anaemia. Anaemia is not physiological if the Hb is less than 10 g/100 ml. The blood picture returns to normal about 6 weeks after delivery. No symptoms and no treatment.

2. IRON DEFICIENCY ANAEMIA

It is the most common cause of anaemia during pregnancy, and is responsible for more than 90% of cases.

Aetiology

1. Fetal demands for iron.
2. Increased production of maternal red blood cells.
3. Iron deficiency in the diet.
4. Vomiting with pregnancy.
5. Hypochlorhydria which is frequent during pregnancy and leads to deficient absorption of iron. Also concurrent antacid use.
6. Multiple pregnancy which increases the iron requirement.

Diagnosis

(1) Complete blood count shows normocytic, normochromic anaemia which becomes microcytic, hypochromic when iron stores are depleted; (2) low serum iron; (3) low serum ferritin; (4) increased total iron-binding capacity. However, this is elevated in normal pregnancy and in absence of iron deficiency, as oestrogen increases all the binding globulins.

Treatment

1. Diet rich in iron, vitamins and proteins.
2. Iron is given by mouth as ferrous sulphate or gluconate, 1 tablet *tds* between meals as food interferes with absorption of iron.
3. If severe anaemia (Hb less than 6 g%) is detected near term (after 36 weeks) the haemoglobin is raised rapidly by intramuscular or intravenous iron preparation (Imferon) or even by packed red cells. Also iron is given parenterally if there is intolerance or no response to oral therapy. One unit of packed red cells increases Hb level by 1 g/dl.

Notes:

- The maximum response of intravenous iron therapy takes 6 weeks.
- Iron tablets should be continued for at least 3 months and better for 6 months after correction of anaemia to replenish the iron store.
- With oral iron therapy the Hb level should increase by at least 0.3 gm per week if the patient is responding.
- The first effect of iron deficiency anaemia is low serum ferritin (less than 15 micrograms per litre) as it indicates reduced amount of stored iron.

3. MEGALOBlastic ANAEMIA OF PREGNANCY

It occurs only in the last trimester and disappears after delivery.

Aetiology

It is due to folate deficiency caused by:

1. Fetal demands for folate.
2. Diminished intake of the vitamin due to loss of appetite or vomiting.
3. Increase in the rate of destruction and excretion of folate which normally occurs in the second and third trimesters of pregnancy.

Diagnosis

1. Symptoms of anaemia as headache, dyspnoea, and palpitation.
2. In severe cases the spleen and liver are enlarged.
3. Complete blood count shows macrocytic, normochromic anaemia. The neutrophils are hypersegmented (>5 lobes). Megaloblasts are present in the bone marrow. Serum iron is high and serum folate is low.

Treatment

Folic acid by mouth, 5 mg daily, if there is vomiting it is given intramuscularly. Diet rich in proteins. Packed red cells for severe cases near term.

Note: The megaloblasts are large erythroblasts with large immature nuclei.

4. ADDISONIAN PERNICIOUS ANAEMIA

Rarely occurs in association with pregnancy. Differs from megaloblastic anaemia of pregnancy in that it is present before pregnancy, it does not disappear after delivery with the presence of achlorhydria and sometimes nervous manifestations. Treated by injection of vitamin B12.

5. APLASTIC ANAEMIA

Due to inhibition of the bone marrow by chemicals as arsenical compounds and chloramphenicol or the use of radioactive agents, sometimes it is idiopathic (50%). Lack of red cells causes hypoxia, of leucocytes predisposes to infection and of platelets leads to haemorrhage. Treated by removal of the cause if found, corticosteroids and repeated blood transfusion until the bone marrow regenerates.

6. SECONDARY ANAEMIA

Secondary to chronic diseases as tuberculosis and chronic nephritis. It is normochromic normocytic in the early stages then becomes hypochromic microcytic. Treat the cause and give iron preparations.

7. HAEMORRHAGIC ANAEMIA

Due to haemorrhage which may be acute as in case of ante- or postpartum haemorrhage or chronic such as due to piles.

8. HAEMOLYTIC ANAEMIA

It may be congenital or acquired caused by incompatible blood transfusion or *Clostridium welchii* infection.

N.B.: Pallor is not a reliable feature of anaemia during pregnancy because of peripheral vasodilatation, so it is essential that the Hb value should be determined in every pregnant patient at the first visit and again at 28 and 36 weeks and more frequent if patient is anaemic.

EFFECT OF ANAEMIA ON PREGNANCY AND LABOUR

If anaemia is severe (Hb below 8 g%); the following complications are liable to occur:

- I. **Maternal complications.** (1) Abortion; (2) preterm labour; (3) uterine atony during labour; (4) retained placenta; (5) postpartum haemorrhage; (6) obstetric shock; (7) puerperal sepsis; (8) venous thrombosis; (9) defective lactation; (10) subinvolution of uterus.
- II. **Fetal complications.** (1) Intrauterine growth restriction; (2) intrauterine death; (3) congenital fetal malformation. These complications are secondary to maternal hypoxia which leads to fetal hypoxia.

N.B.: The fetus obtains his needs of iron even if the mother is iron-deficient.

PYELONEPHRITIS OF PREGNANCY

INCIDENCE

It occurs in 1–2% of pregnancies.

AETIOLOGY

1. *The predisposing factor* is stasis of urine in the renal pelvis which is due to:
 - a) Pressure on the ureters by the pregnant uterus against the pelvic brim. Therefore pyelonephritis is more common on the right side, due to right obliquity of the uterus, and pressure of the dilated right ovarian vein and is more in primigravidae, due to tightness of the anterior abdominal wall. The left ureter is protected from pressure by the sigmoid colon.
 - b) Atony and dilatation of the ureter due to progesterone which causes relaxation of the smooth muscle fibres.
 - c) Hypertrophy of the wall of the pelvic portion of the ureter leading to narrowing of the lumen, but this may be a defensive mechanism against pressure by the head in the pelvis.
 - d) The presence of ureteric stone or stricture, which may be congenital or acquired.
2. *Causative organisms*: *Escherichia* (E.) *coli* is the commonest organism (over 80%). Occasionally infection is caused by other organisms as staphylococci or streptococci. Mixed infection may occur.
3. *Routes of infection*
 - a) Bloodstream from septic focus, e.g. tonsils, teeth, etc.
 - b) Ascending infection from the bladder along the periureteric lymphatics or along the lumen of the ureters as the result of vesico-ureteral reflux. Organisms present in the urethra may enter the bladder by pressure on the urethra during coitus and by catheterization. Ascending infection is the commonest route.
 - c) Spread from the colon by lymphatics.

PATHOLOGY

1. *Kidney*: The renal pelvis is dilated. Its wall is congested, oedematous, with leucocytic infiltration, and may be ulcerated. The renal tissue shows a variable degree of inflammatory reaction.
2. *Ureter*: It is dilated, elongated and tortuous.
3. *Bladder*: Normal or may show cystitis as a result or a cause of pyelonephritis.

DIAGNOSIS

The condition usually occurs in the second trimester after the 16th week occasionally (20%) it occurs 6–10 days after delivery due to catheterization of bladder during labour. It

is more common on the right side and more in primigravidae. There are 2 main clinical types:

A) Acute Pyelonephritis

Symptoms

1. Sudden onset of severe pain in one or both loins. It usually radiates along the course of the ureter to the suprapubic region with painful and frequent micturition (cystitis).
2. General symptoms of infection in the form of fever, headache, nausea, vomiting and rigors.

Signs

1. *General*: Fever and rapid pulse.
2. *Abdominal*: Tenderness and rigidity in one or both renal angles.

INVESTIGATIONS

1. Urine analysis: There is proteinuria, many pus cells and bacteria. Infection with *E. coli* makes the urine acidic in reaction with a fishy odour.
2. Urine culture and sensitivity test.
3. Complete blood count. It shows leucocytosis.
4. Blood urea, serum creatinine and serum electrolytes to assess kidney function.

B) Chronic Pyelonephritis

This may take the form of dull aching loin pain with painful and frequent micturition, backache, gastrointestinal disturbances, unexplained pyrexia or rigors.

The renal angle and the suprapubic region may be tender. The urine contains protein, pus cells and organisms.

DIFFERENTIAL DIAGNOSIS

1. Acute type: Has to be differentiated from causes of acute abdomen as acute appendicitis, acute cholecystitis, etc. If vomiting is marked it may be diagnosed as hyperemesis gravidarum.
2. Chronic type: Has to be differentiated from chronic appendicitis and myositis.

COMPLICATIONS

1. Acute type: (a) Septic shock (systemic inflammatory response syndrome); (b) adult respiratory distress syndrome.
2. Chronic type: (a) Renal hypertension; (b) complications during pregnancy (see chronic renal disease with pregnancy).

TREATMENT

1. General Treatment

- a) Patient is hospitalized to receive intravenous antibiotic therapy and to monitor urine output.
- b) Rest in bed on the healthy side to avoid pressure of the uterus on the affected ureter to help drainage.
- c) Light diet and excessive fluids (at least 3 litres daily).
- d) Symptomatic treatment; analgesics for pain, antipyretics, a mild laxative to open the bowels.
- e) An alkali as potassium citrate is given orally to make urine alkaline. *E. coli* does not flourish in alkaline medium.

2. Antibiotic Therapy

Urine is sent for culture and sensitivity test. We start with one of the cephalosporin antibiotics as many cases of *E. coli* infection (33%) are resistant to ampicillin. The antibiotic is given intravenously and continued until the patient is afebrile for at least 48 hours then oral antibiotic therapy is given for at least two weeks. The antibiotic may be changed according to the clinical response and the result of urine sensitivity test. Patient is considered cured if three successive urine cultures taken at weekly interval are negative. Culture is then repeated monthly until delivery.

Postpartum Follow Up

In case of recurrent pyelonephritis, sonography is carried out during pregnancy to exclude urinary tract abnormalities as stone. Also intravenous pyelography is performed at least 12 weeks after delivery to allow the pregnancy related anatomical changes to resolve.

ASYMPTOMATIC BACTERIURIA

Bacteriuria is the mere presence of bacteria in urine without pus cells. It occurs in 5–10% of pregnant women. Bacteriuria is significant if the number of organisms (a single pathogen) equals or exceeds 100,000 per ml of urine on two occasions. This condition predisposes to cystitis, acute pyelonephritis (30%), anaemia, hypertension, preterm labour and intrauterine growth restriction. It is wise to culture urine at the first antenatal visit and if significant bacteriuria is found it should be treated. An antibiotic as cephalexin (Keflex) 500 mg orally 6 hourly for ten days or nitrofurantoin 100 mg orally 6 hourly for ten days. The patient is followed by repeated urine culture as mentioned before as asymptomatic bacteriuria is liable to recur (30%).

Note: Patients with recurrent urinary tract infection should receive a suppressing dose of antibiotic for the duration of pregnancy and the first two weeks postpartum, e.g., cephalexin (Keflex) 500 mg tablet or nitrofurantoin 100 mg tablet once daily at bed time (to have a high concentration of the drug in the bladder for several hours) or the dose of antibiotic is given only after each coitus. Also the woman is asked to pass urine immediately after coitus. Reinfection is thought to arise from fecal bacteria coming from the anus and contaminating the urethra. Pressure on the urethra during coitus (milking of the urethra) may force the organisms to enter the bladder.

DIABETES MELLITUS WITH PREGNANCY

INCIDENCE

2–12% of pregnancies are complicated by diabetes mellitus, 90% are gestational diabetes, and 10% had diabetes mellitus before pregnancy (pregestational or preconceptional diabetes mellitus).

CLASSIFICATION

1. *Clinical or overt diabetes*: Abnormal glucose tolerance test (GTT) associated with symptoms or signs of diabetes. Symptoms of diabetes include polyuria, polyphagia, polydipsia, pruritus and symptoms of peripheral neuritis.
2. *Subclinical or chemical diabetes*: Abnormal GTT but no symptoms or signs of diabetes. Hormonal contraceptives may cause chemical diabetes.
3. *Gestational diabetes*: The woman becomes diabetic during pregnancy because of the hormonal changes. Most of the cases become normal after delivery but some remain diabetic.
4. *Potential diabetes*: Normal GTT but the woman has a risk factor. Risk factors include family history of diabetes in a first degree relative, obesity, delivery of large babies (4 kg or more), previous malformed babies and previous unexplained perinatal deaths. The potentially diabetic is more liable to develop diabetes at some time during her life.

DIABETOGENIC EFFECT OF PREGNANCY

Diabetes may appear only during pregnancy or become aggravated by pregnancy because the human placental lactogen, prolactin, oestrogens, progesterone and cortisol antagonise insulin (reduce the number and sensitivity of insulin receptors in target cells). Also the placental enzyme insulinase destroys insulin and may play a role.

EFFECT OF PREGNANCY ON DIABETES

1. The disease may appear for the first time during pregnancy.
2. The insulin requirement gradually increases after the third month until term, so ketosis readily occurs. However, during the early months of pregnancy the amount of insulin needed may be reduced because nausea and vomiting lead to maternal hypoglycaemia, so diabetes becomes difficult to control in pregnancy.
3. Diabetic nephropathy, retinopathy and neuropathy may be aggravated in pregnancy. Diabetic retinopathy will progress in more than 50% of pregnant diabetics. The cause is unknown but may be due to human placental lactogen.
4. During labour there is liability to hypoglycaemia because of uterine activity.

5. After delivery the insulin requirement decreases due to drop in the level of placental hormones which antagonise insulin.

EFFECT OF DIABETES ON PREGNANCY

A) Fetal Complications

1. Intrauterine death occurs in about 5% of cases especially after 36 weeks. It may be due to maternal ketosis, hypoglycaemia, congenital malformation, associated pre-eclampsia or placental insufficiency (the fetus may outgrow its placenta or due to atherosclerosis of pelvic arteries).
2. Neonatal death (5%) due to prematurity, respiratory distress syndrome, congenital anomalies, and hypoglycaemia resulting from fetal hyperinsulinaemia.
3. Increased incidence of congenital malformation (5–10%). It is related to maternal hyperglycaemia during the period of organogenesis. All organs can be affected. Commonest lesion is cardiac anomalies. Absence of sacrum (sacral agenesis or caudal regression syndrome) is specific.
4. Macrosomia : The infant is often long, large and fatty weighing 4 or more kilograms. It is related to maternal hyperglycaemia which leads to fetal hyperglycaemia and fetal hyperinsulinaemia. Insulin is a growth hormone and in the presence of hyperglycaemia leads to more formation of glycogen, fat and protein. There is increase in body fat, muscle mass and organomegaly (except brain and kidneys). Like the fetus the placenta and umbilical cord increase in size. Macrosomia complicates 25–50% of cases.
5. Intrauterine growth restriction which is less frequent. It occurs when diabetes is complicated by vascular disease leading to atherosclerosis of pelvic arteries and placental insufficiency (class D and above).
6. Fetal birth injuries due to macrosomia.
7. The infant may inherit diabetes which may appear later in life in about 1% while the risk is 1 in 1000 in the general population.

Note: Caudal regression syndrome means absent sacrum with hypoplasia of lower limbs.

B) Maternal Complications

1. Abortion if the disease is not controlled.
2. Preterm labour.
3. Increased incidence of pre-eclampsia (30%).
4. Increased incidence of polyhydramnios (30%).
5. Increased liability to infection as urinary tract infection, puerperal sepsis and vulvo-vaginal mycosis.
6. Obstructed labour due to large baby.

7. Postpartum haemorrhage is more common.
8. Defective lactation.
9. Difficulty in control of diabetes leading to hyperglycaemic or hypoglycaemic coma.

MANAGEMENT

A) Before Pregnancy

1. Good control of blood glucose level by control of diet, daily exercise and treatment.
2. The diabetic woman should take folic acid 5 mg daily before conception and for the first 12 weeks of pregnancy to reduce the incidence of neural tube defects.
3. Early booking for antenatal care when pregnancy occurs.

B) During Pregnancy

1. Control of diabetes by diet, daily exercise and insulin if necessary. Oral hypoglycaemic agents are not used because they do not control properly the glucose level, may cause severe hypoglycaemia in the newborn and may be teratogenic. Diet should supply 30–35 Kcal/kg of ideal body weight (50% carbohydrates, 20% proteins, 30% fats).
2. The case should be seen by the obstetrician, diabetitian and dietitian.
3. A screening test is done routinely for all pregnant women. The test is done at 24–28 weeks. If abnormal, a GTT using 100 g oral glucose is carried out (one of the screening tests is to give 50 g oral glucose and if the venous plasma glucose at 1 hour is 140 mg% or more, the test is abnormal).
4. Measurement of maternal serum alpha-fetoprotein at 16–18 weeks to diagnose neural tube defects (high level). Abnormal result is confirmed by sonography.
5. Sonography: (a) In the first trimester to confirm gestational age; (b) at 16–18 weeks to exclude congenital fetal anomalies; (c) echocardiogram at 20–24 weeks to exclude congenital heart disease; (d) repeated scans every 1–2 weeks if there is intrauterine growth restriction or macrosomia.
6. Estimation of glycohaemoglobin (glycosylated haemoglobin - HbA1c), to provide information about diabetic control. If level is high it means hyperglycaemia has been present for at least 1–2 months. The test is done monthly or glycosylated plasma proteins are measured every 2 weeks.
7. Examination of fundus oculi for diabetic retinopathy. It is performed once every trimester, and more frequently if there is retinopathy.
8. The patient is seen every 2 weeks till 28 weeks and then weekly.
9. Admission to hospital is indicated if diabetes is not controlled or complicated, e.g. by pre-eclampsia.

10. Measures to assess fetal well-being. Daily kick count and nonstress test done twice weekly or daily in severe cases (testing starts at 28–30 weeks).
11. If diabetes is well controlled pregnancy is terminated at 38–40 weeks. If for any reason pregnancy needs to be terminated before 38 weeks amniocentesis is done to determine lung maturity. The presence of phosphatidylglycerol (more than 2 mg%) is more reliable than the L/S ratio (lecithin/sphingomyelin ratio) which must be 3.5 in diabetic cases. If there is no evidence of lung maturity, dexamethasone is given for 24 hours (12 mg IM every 12 hours for 2 doses) then pregnancy is terminated.

Methods of Termination of Pregnancy

Artificial rupture of membranes and oxytocin drip or caesarean section. The first method is applied when the fetus is not large, vertex presentation and favourable cervix, otherwise caesarean section is done. Prostaglandin vaginal tablet or gel may be used to ripen the cervix and to induce labour.

Notes:

- Fetal hyperinsulinaemia inhibits formation of lung surfactant and delays lung maturity.
- Therapeutic abortion may be indicated if there is diabetic retinopathy or nephropathy (Kimmelstiel Wilson syndrome), or ischaemic heart disease. However, these are not absolute indications but the risks should be explained to the patient.
- A screening test should be done routinely because about 50% of cases of gestational diabetes are asymptomatic, and in 50% of cases there is no risk factor.
- When GTT with 100 gm glucose is performed during pregnancy, the patient is diagnosed diabetic when 2 or more venous plasma values equal or exceed the following: fasting, 105 mg/dl; 1h, 190 mg/dl; 2h, 165 mg/dl and 3h, 145 mg/dl.

C) During Labour

1. On the morning of induction a continuous infusion of insulin in glucose is given if the patient was on insulin. Ten units in 500 ml of 5% glucose is given at a rate of 125 ml/hour (2.5 units per hour).
2. Blood glucose is determined every 2 hours.
3. Urine is checked for ketones every 2 hours.
4. Continuous monitoring of the fetal heart rate.

D) During the Puerperium

After delivery the drop in the level of placental hormones reduces the dose of insulin needed, so the patient is given one-third to one-half her dose of insulin. Random blood glucose level of 150 to 200 mg/dl is acceptable.

E) The Newborn

It is managed as a preterm baby irrespective of its weight. It is liable to develop respiratory distress syndrome (RDS), hypoglycaemia, hypocalcaemia (tetany), hypo-

magnesaemia, polycythaemia (due to intrauterine hypoxia or hyperglycaemia stimulates erythropoietin production), hyperbilirubinaemia (due to polycythaemia and increased red cell destruction) and cardiomyopathy. RDS is the commonest cause of death in the neonatal period (50–75%). Cardiomyopathy is due to deposition of glycogen in the myocardium and it leads to fetal oedema.

Notes:

1. Insulin does not cross the placenta.
2. Neonatal hypocalcaemia (serum level <7 mg/dl) may occur. It is due to failure of synthesis of parathyroid hormone, the cause of which is unknown.
3. Neonatal hypoglycaemia means a blood glucose level below 40 mg/dl. Maternal blood glucose levels greater than 90 mg/dl during labour increases the risk of neonatal hypoglycaemia.
4. The dose of insulin depends upon the ideal body weight, and duration of pregnancy in weeks. In the first trimester the dose is 0.5 to 0.6 U/kg of ideal body weight; in the second trimester: 0.7 to 0.8 U/kg; and in the third trimester: 0.9 to 1.0 U/kg. However, this guideline has to be modified to achieve euglycaemia. Two-thirds of the dose is given in the morning: 2/3 intermediate acting + 1/3 rapid acting insulin. One-third of the dose is given in the evening: 1/2 intermediate acting + 1/2 rapid acting insulin. The new trend is to give four injections daily to ensure better control of glucose level most of the time to reduce complications. A long or intermediate acting insulin is given once daily, and a short acting insulin is given three times daily pre-prandially.
6. Diet. Small frequent meals (6) are given 2–3 hours apart to avoid ketosis: breakfast; morning snack; lunch; afternoon snack; dinner; and bedtime snack. The latter prevents hypoglycaemia during the night which may cause rebound hyperglycaemia in the morning (Somogyi phenomenon).
6. The normal value of HbA1c is 4–6% of the total haemoglobin. It should be kept below 8%.
7. The hyperglycaemia of gestational diabetes (GD) usually starts in the second half of pregnancy.
8. GD recurs in more than 50% of cases in a subsequent pregnancy.
9. More than 50% of women with GD will develop clinical (overt) diabetes within 20 years if they remain obese.
10. Patients with GD should have a GTT (with 75 g) done 6 weeks after delivery.

Contraception

The mechanical methods (condom or diaphragm) and sterilization (male or female) are the best. The combined hormonal contraceptive pill is contraindicated as it increases the hyperglycaemia and diabetes and also increases the risk of myocardial infarction and thromboembolism in the diabetic woman. However, the progestin-only pill and the triphasic pill can be used as they do not produce significant metabolic changes in the diabetic woman. The intrauterine device increases the risk of pelvic infection in diabetic woman and better avoided. However, no method of contraception is absolutely contraindicated for the diabetic woman.

PHYSIOLOGICAL GLYCOSURIA OF PREGNANCY

Some healthy women (about 15%) excrete a small amount of glucose in urine during pregnancy. It is due to increased glomerular filtration rate which occurs normally during pregnancy and inability of the renal tubules to reabsorb the extra amount of filtered glucose (impaired renal threshold). To avoid the possibility of physiological glycosuria, the

pregnant woman should be fasting before testing her urine. This is the commonest cause of glycosuria during pregnancy. Because of physiological glycosuria urine testing has no place in the control of diabetes in pregnancy as it does not reflect the blood glucose level accurately.

REDUCING SUBSTANCES IN THE URINE OF PREGNANT WOMEN

1. Glucose
 - a) Physiological glycosuria of pregnancy.
 - b) Diabetes mellitus.
2. Lactose: Lactosuria may occur during late pregnancy, labour or lactation due to activity of the breasts. Differentiation from glycosuria is by the use of glucose strips as the Tes-Tape or Diastix which identifies glucose.
3. Vitamin C, salicylates and morphine.

THYROID DISORDERS IN PREGNANCY

HYPERTHYROIDISM

Maternal thyroid function is increased in normal pregnancy due to increased production of the thyroid stimulating hormone (TSH) from the anterior pituitary, and due to effect of chorionic thyrotrophin secreted by the placenta (trophoblast). Also, human chorionic gonadotrophin has a thyroid stimulating effect. Normal pregnancy may lead to mild thyroid enlargement, tachycardia, palpitation, and warm extremities. For this reason, mild hyperthyroidism is difficult to diagnose during pregnancy, and the diagnosis depends upon raised free thyroxine (FT4) or its equivalent free thyroxine index (FT4I). The total serum T4 is normally increased in pregnancy as oestrogen increases the thyroxine-binding globulin.

Incidence

About 1 in 1000 pregnancies.

Aetiology

- 1) Graves disease is the most common cause of hyperthyroidism (thyrotoxicosis) in pregnancy (85% of cases).
- 2) Toxic multinodular goitre.
- 3) Toxic adenoma (single toxic nodule).
- 4) Acute (subacute) thyroiditis, probably due to viral infection. The virus causes damage of the follicles and release of excess hormone.
- 5) Hydatidiform mole due to chorionic thyrotrophin secreted by the trophoblast, also human chorionic gonadotrophin has a thyroid stimulating effect (it stimulates the TSH receptors on the thyroid gland).
- 6) Struma ovarii which is an ovarian teratoma composed mainly of thyroid tissue, which may hyperfunction (5%).

Complications

A) Maternal

There is increased risk of abortion, preterm labour, pre-eclampsia, and heart failure. Mild hyperthyroidism may cause hyperemesis gravidarum. Infertility may occur due to anovulation.

B) Fetal

It may lead to intrauterine growth restriction and intrauterine death. Graves disease may lead to fetal tachycardia, neonatal hyperthyroidism and enlarged thyroid due to the

passage of autoantibodies across the placenta. These stimulate the fetal thyroid gland leading to thyrotoxicosis and goitre in less than 8% of cases. These antibodies are known as long acting thyroid stimulating antibodies (LATS) or TSH receptor antibodies (have same action as TSH).

Note: TSH does not cross the placenta. T3 and T4 are inactivated in the placenta by deiodinase enzyme, and so very small amounts reach the fetus.

Treatment

1. **Antithyroid drugs.** They reduce the formation of thyroid hormones and include propylthiouracil (PTU), carbimazole, and methimazole (the active metabolite of carbimazole). PTU is the drug of choice in pregnancy as carbimazole and methimazole have been associated with aplasia cutis in the newborn (rare). The dose of PTU is 200-400 mg daily given in 2 or 3 divided doses. The dose of methimazole is 20-40 mg daily given in 2 or 3 divided doses (one tenth the dose of PTU). The antithyroid drugs cross the placenta leading to fetal hypothyroidism and goitre. The drugs are excreted in milk so breast-feeding is contraindicated. However, if PTU is used in a small dose less than 200 mg daily breast-feeding is allowed. The drug is given immediately after infant suckling 4 times daily, and the infant is followed with thyroid function tests.
2. **Beta-blockers** as atenolol (Tenormin). The drug causes rapid control of thyrotoxic symptoms, and can be combined with antithyroid drugs until they become fully effective then the drug is stopped. The drug blocks the beta-adrenergic receptors thus prevents adrenergic symptoms of thyrotoxicosis and also blocks conversion of T4 to T3. Dose is 50-100 mg daily. Avoided in asthma and given with care in heart failure.
3. **Partial thyroidectomy.** Indications are: (a) failure of medical treatment, (b) toxic reactions to the drug, (c) large goitre causing obstructive symptoms.

HYPOTHYROIDISM

Incidence

About 3-9 per 1000 pregnancies.

Aetiology

A) *Primary hypothyroidism*

It is due to a disease of the thyroid gland, and so TSH is high. Causes are:

1. Hashimoto thyroiditis. Commonest cause. It is an autoimmune thyroiditis.
2. Iodine deficiency.
3. Gland ablation by surgery, or treatment with radioactive iodine for Graves disease.
4. Antithyroid drug therapy.

5. Ingestion of goitrogens, e.g., thiocyanate and lithium.

B) Secondary hypothyroidism

It is secondary to hypothalamic or pituitary disease, as in case of Sheehan syndrome, and chromophobe adenoma of pituitary gland. TSH is low.

Complications

A) Maternal

There is increased risk of abortion, preterm labour, and pre-eclampsia. Infertility may occur due to anovulation.

B) Fetal

It may lead to intrauterine growth restriction, and intrauterine death.

Treatment

Replacement therapy with thyroxine (T₄). It is converted in the body to T₃ which is the active thyroid hormone. The dose of levothyroxine is 50-200 micrograms/day. Desiccated thyroid extract which is a mixture of T₄ and T₃ can be used. Breast-feeding is not contraindicated.

Notes:

- Hashimoto thyroiditis may be associated with normal thyroid function, or hyperthyroidism, or hypothyroidism in longstanding cases.
- Subclinical hypothyroidism means elevated TSH with absent symptoms. The levels of T₃ and T₄ are normal.

POSTPARTUM THYROIDITIS

It is an autoimmune disorder of unknown aetiology. It occurs after delivery in 2-5% of cases. The patient presents with hyperthyroidism which occurs 3 to 6 months after delivery. This is followed by a hypothyroid state which is usually transient lasting up to 3 months. Thyrotoxicosis results from excessive release of thyroid hormones from destroyed follicles and not from increased production of hormones. For this reason antithyroid drugs as PTU are not effective. The treatment is by a beta-blocker as atenolol to block the sympathetic symptoms of thyrotoxicosis. There is increased risk of recurrence with subsequent deliveries. There is also an increased risk of developing Graves disease or Hashimoto thyroiditis.

ANTIPHOSPHOLIPID ANTIBODY SYNDROME (APS)

The syndrome is characterized by: (1) thrombosis (arterial and venous), (2) recurrent fetal loss, (3) thrombocytopenia, (4) positive laboratory tests for antiphospholipid antibodies as anticardiolipin antibodies and lupus anticoagulant. The syndrome may occur alone (primary APS), or in association with systemic lupus erythematosus or other connective tissue diseases (secondary APS).

EFFECTS OF ANTIPHOSPHOLIPID ANTIBODIES ON PREGNANCY

1. Recurrent abortion. Typically after 10th week of pregnancy.
2. Intrauterine growth restriction.
3. Intrauterine death.
4. Pre-eclampsia which appears before 20 weeks of pregnancy and it is usually severe.
5. Placental abruption.
6. Preterm labour.

They may also cause infertility by preventing implantation of the blastocyst.

DIAGNOSIS

Antiphospholipid antibodies appear in response to infection, malignant disease, drugs, and stress, and are present in low titres in about 2% of normal persons. For diagnosis high titres should be used and tests repeated after 3 months. Only patients with persistently positive high titres associated with clinical features of the syndrome should be treated.

TREATMENT

A small dose of aspirin 75 mg tablet is given daily combined with heparin 5000 units subcutaneously twice daily or low-molecular weight heparin once or twice daily, and its dose depends upon the preparation used. This dose of heparin, and the low-molecular weight heparin do not prolong the activated partial thromboplastin time and so do not need laboratory monitoring of the treatment. If this therapy fails immunoglobulin infusion is given (IgG).

SYPHILIS IN PREGNANCY

EFFECT OF SYPHILIS ON PREGNANCY

1. Abortion. The spirochetes cross the placenta in a significant number after the twentieth week of gestation, when the Langhans layer starts to disappear. So fetal loss due to syphilis does not occur before the 5th month and so syphilis is no more considered as a cause of abortion.
2. Intrauterine fetal death.
3. Neonatal death.
4. Congenital syphilis.

The syphilitic mother can transmit infection to the fetus at any stage of the disease. However, if the duration of syphilis is less than one year the fetus will be affected in almost 100% of cases. As the years pass from the time of infection the likelihood of the fetus to be affected diminishes and after 5 years the mother would probably produce a healthy baby whether she is treated or not.

EFFECT OF PREGNANCY ON SYPHILIS

Pregnancy appears to decrease the severity of syphilis, so if infection occurs during pregnancy the chancre of primary syphilis is often masked or minimal.

DIAGNOSIS

1. History of contracting the disease.
2. History of intrauterine or neonatal death.
3. Presence of characteristic clinical signs of syphilis.
4. *Treponema pallidum* can be identified in smears taken from overt lesions as the chancre using the dark ground microscope.
5. Positive serology. Every pregnant woman should have a screening test as the WR (Wassermann Reaction) or VDRL (Venereal Disease Research Laboratory Test). If the test is positive a specific treponemal test is done as the *Treponema Pallidum* Immobilization Test (TPI) or the Fluorescent *Treponemal* Antibody Absorption Test (FTA-Abs) to confirm the diagnosis.
6. Examination of newborn for evidence of congenital syphilis as saddle nose, etc.
7. Examination of the placenta. It is large, heavy, pale, yellowish grey and non friable. Microscopically the villi are large and club-shaped. The vessels are reduced in number as they are obliterated by endarteritis. Spirochetes may be detected in the villous stroma.

TREATMENT

It is the same for the pregnant and nonpregnant woman and depends upon the clinical stage of the disease.

I) Primary and Secondary Syphilis

- a) Procaine penicillin 600,000 units IM daily for 10 days or the long acting Benzathine penicillin 2.4 million units IM (half the dose in each buttock).
- b) For patients allergic to penicillin; oral erythromycin or cephalosporin 500 mg 4 times daily for 15 days.

II) All Other Syphilis (Latent, gummatous, neuro and cardiovascular)

- a) Procaine penicillin 600,000 units IM daily for 20 days or Benzathine penicillin 2.4 million units IM weekly for 3 weeks. The long acting penicillin is contraindicated in neurosyphilis because it does not reach enough concentration in the cerebrospinal fluid.
- b) For patients allergic to penicillin; oral erythromycin or cephalosporin 500 mg 4 times daily for 30 days.

Notes:

- Erythromycin does not cross the placenta freely and so if the drug is given to the mother the newborn should be treated with penicillin immediately after delivery even if the baby is completely normal.
- Several factors can cause a false positive nontreponemal screening test such as bacterial and viral infections, and chronic liver disease. Accordingly a positive screening test must be confirmed by a specific treponemal assay.

FIBROIDS WITH PREGNANCY

INCIDENCE

About 1%. Most of the patients are above 30 years.

EFFECT OF FIBROIDS ON PREGNANCY

1. Fibroids may cause infertility.
2. Abortion (usual with submucous, sometimes with interstitial and rare with subserous fibroid).
3. Posterior wall fibroid may cause incarceration of a retroverted gravid uterus.
4. Preterm labour.
5. Pressure symptoms:
 - a) Large abdominal tumour may lead to abdominal discomfort, dyspnoea and palpitation.
 - b) Fibroid in the pelvis may lead to pressure on the bladder, rectum and pelvic veins.
6. Malpresentation.
7. Nonengagement of the presenting part.
8. Ectopic pregnancy due to obstruction of the tube.
9. Placenta praevia, due to interference with implantation of the ovum in the upper part of the uterus.
10. Acute abdomen; due to torsion of a pedunculated subserous fibroid, red degeneration or internal haemorrhage due to rupture of a vein on the surface of a subserous fibroid.

EFFECT OF FIBROIDS ON LABOUR

1. Obstructed labour, due to a cervical fibroid or a subserous fibroid impacted in the pelvis below the presenting part.
2. The presence of fibroids mechanically interferes with uterine contractions leading to atony. Uterine atony leads to prolonged labour, retained placenta and postpartum haemorrhage.
3. Submucous fibroid predisposes to placenta accreta.



Fig. 38. Fibroid obstructing labour

EFFECT OF FIBROIDS ON PUERPERIUM

1. Subinvolution of uterus.
2. Secondary postpartum haemorrhage (caused by a submucous fibroid or fibroid polyp).
3. Inversion of uterus may be caused by a fundal submucous fibroid.

4. Increased incidence of puerperal sepsis due to infection of a traumatized tumour and interference with drainage of the uterus.

EFFECT OF PREGNANCY ON FIBROIDS

1. Increase in size, due to increased vascularity and oedema and hypertrophy of muscle fibres. After delivery the tumour returns to its original size.
2. Softening due to increased vascularity and oedema and so it may be difficult to detect the tumour by palpation.
3. Red degeneration may occur during pregnancy, labour or puerperium (10%).
4. Intraperitoneal haemorrhage may occur due to rupture of a vein on the surface of a subserous tumour.
5. A pedunculated subserous fibroid may undergo torsion.
6. A submucous fibroid may become extruded during the puerperium leading to haemorrhage. The tumour may be detached spontaneously.
7. The tumour may become traumatized during labour and this predisposes to infection.

MANAGEMENT

A) During Pregnancy

No interference, however, myomectomy is performed in the following conditions:

1. Red degeneration which does not respond to medical treatment or when the diagnosis is doubted. Only the affected tumour is removed (known by adherent omentum).
2. Torsion of the pedicle of a subserous fibroid.
3. Haemorrhage from the surface of a subserous tumour.

B) During Labour

If a fibroid causes obstruction to labour the fetus is delivered by caesarean section. Myomectomy is not done at the same time due to the danger of severe haemorrhage unless the fibroid is subserous with a stalk. Caesarean hysterectomy is permissible in a patient with multiple fibroids over 40 years. Fibroids detected during the puerperium need no treatment except after 3–6 months.

OVARIAN TUMOURS WITH PREGNANCY

INCIDENCE

1 in 1,000 cases. Dermoid cyst is frequent and accounts for 25% of cases because the cyst occurs at young age (20–30 years). Mucinous cystadenoma also accounts for 25% of cases.

EFFECT OF OVARIAN TUMOURS ON PREGNANCY

1. Abortion.
2. Preterm labour.
3. Pressure symptoms:
 - a) Large abdominal tumour may lead to abdominal discomfort, dyspnoea and palpitation.
 - b) A tumour in the pelvis may lead to pressure on the bladder, rectum and pelvic veins.
4. Malpresentation.
5. Nonengagement of the presenting part.
6. Obstructed labour when the tumour is impacted below the presenting part.

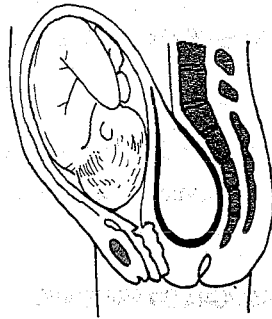


Fig. 39. Incarcerated ovarian cyst obstructing labour.

EFFECT OF PREGNANCY ON OVARIAN TUMOURS

The following complications are more liable to occur during pregnancy; torsion, haemorrhage, rupture, infection and rapid growth.

MANAGEMENT

A) During Pregnancy

With a small ovarian cyst less than 5 cm in diameter the patient is kept under observation as the cyst is most probably functional, e.g., corpus luteum cyst. However, if the cyst is 5 cm or more it must be removed because it is considered a neoplastic cyst and we cannot be sure that it is not malignant or it will turn malignant and to avoid complications as torsion. Also if the tumour is solid it must be removed. Operation is done in the second trimester of pregnancy (between 13 and 28 weeks). Operation is not done in the first trimester as the corpus luteum of pregnancy may be injured leading to abortion. Operation is also not done in the third trimester (after 28 weeks) to avoid preterm labour and technical difficulty, but the tumour should be removed within one week after delivery to avoid complications, especially torsion.

B) During Labour

1. If the tumour is lying above the pelvic brim and not causing obstruction: Vaginal delivery is allowed and the tumour is removed within the first week of puerperium.
2. If the tumour is below the presenting part and causing obstruction caesarean section is done and the tumour is removed at the same time.

Ovarian tumour discovered during the puerperium should be immediately removed as torsion is very liable to occur.

INVASIVE CARCINOMA OF THE CERVIX DURING PREGNANCY**INCIDENCE**

1 in 5,000 to 1 in 20,000 pregnancies. It is very rare because the commonest age incidence of carcinoma is 40-50 years, also the presence of infection interferes with pregnancy.

EFFECT OF CARCINOMA ON PREGNANCY AND LABOUR

1. Abortion and preterm labour may occur in advanced cases because of haemorrhage, infection, fever and toxæmia.
2. During labour there is liability to slow dilatation of cervix, obstructed labour, rupture of uterus and cervical lacerations.
3. Puerperal sepsis due to spread of infection from the tumour.

EFFECT OF PREGNANCY AND LABOUR ON CARCINOMA

1. Rapid growth may occur because tumours occurring at young age tend to grow rapidly. The hormonal and vascular changes of pregnancy do not stimulate the rapid growth.
2. The trauma of vaginal delivery may lead to rapid growth and extension of the tumour and so vaginal delivery is contraindicated. Also vaginal delivery can cause severe cervical laceration.

TREATMENT

If the duration of pregnancy is less than 12 weeks the treatment is either radiotherapy in the presence of pregnancy or Wertheim operation with the uterus intact if the case is operable. If the case is seen after the 12th week, it is treated by abdominal hysterotomy or caesarean section followed by radiotherapy when the wound heals (after about 2 weeks) or by immediate Wertheim operation if the case is operable.

N.B.: Radiotherapy in early pregnancy. External irradiation is given. If spontaneous abortion does not occur evacuation is done and later on radium is applied.

RETROVERTED GRAVID UTERUS

FATE (CLINICAL COURSE)

1. *Spontaneous correction or rectification* occurs in most cases.
2. *Abortion* may occur. It is due to congestion of the endometrium and mechanical irritation by sacral promontory. It occurs after the 12th week.
3. *Incarceration*: Rare (1 in 4000 pregnancies), occurs after the 12th week. The body of the uterus remains below the sacral promontory and it continues to grow in the pelvis. It is caused by a projecting promontory, pelvic adhesions or posterior wall fibroid.
4. *Anterior sacculation of the uterus*: Very rare. The fundus remains beneath the sacral promontory and pregnancy continues to grow at the expense of the anterior wall of the uterus. Labour is liable to occur prematurely. Rupture of uterus may occur during pregnancy or labour.

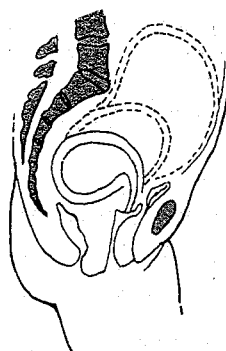


Fig. 40. Anterior sacculation

CLINICAL PICTURE OF INCARCERATION

Symptoms

1. Symptoms of early pregnancy.
2. Urinary symptoms: Frequency of micturition, followed by dysuria and finally acute retention of urine (due to elongation and compression of urethra). If the condition is not recognized retention with overflow occurs.
3. Other symptoms: Pain in lower abdomen due to distension of the bladder, pain in the pelvis due to pressure on pelvic organs, dyspareunia and dyschezia.

Signs

1. *General examination*: Breast signs of pregnancy.
2. *Abdominal examination*: The distended bladder is felt as a cystic tender swelling in the suprapubic region. It may reach the level of the umbilicus. The passage of a urinary catheter evacuates the bladder and reveals the diagnosis.
3. *Vaginal examination*: The cervix is high up behind the symphysis pubis, difficult to reach, the external os is directed forwards or upwards and forwards. The body of the uterus is felt as a soft mass through the posterior vaginal fornix.

DIFFERENTIAL DIAGNOSIS

1. Ovarian cyst impacted in Douglas pouch.
2. Posterior wall fibroid impacted in Douglas pouch.
3. Pelvic haematocele.

TREATMENT

1. If retroversion is discovered during pregnancy, nothing is done because spontaneous correction usually occurs. The patient is advised to keep her bladder empty by frequent micturition and to lie on her abdomen as much as she can. If, however, retroversion is responsible for recurrent abortion, the uterus is corrected at 10th week and a Hodge pessary is left in place till the 14th week (when the uterus has become an abdominal organ).
2. If incarceration occurs: A Foley catheter is inserted to evacuate the bladder and keep it empty. The patient lies on her abdomen as much as she can. She is advised to lie in the knee-chest position for about 15 minutes several times daily. Antibiotics are given to prevent urinary infection. Usually spontaneous correction occurs. If this does not occur after two days, manual correction is tried. If it fails it is done under general anaesthesia and a Hodge pessary is applied. If reposition fails the uterus is evacuated. Abdominal hysterotomy may be needed because vaginal evacuation is difficult.

Note: In anterior sacculation delivery is by caesarean section.

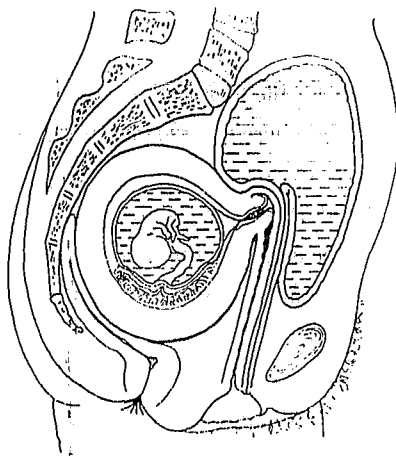


Fig. 40-2. Incarcerated retroverted gravid uterus.

PENDULOUS ABDOMEN

The pregnant uterus falls forwards and overhangs the symphysis pubis.

CAUSES

1. Contracted pelvis.
2. Lax abdominal wall.
3. Grand multipara due to lax abdominal and uterine walls.
4. Divarication of recti muscles.
5. Incisional hernia.
6. Increased lumbar lordosis.

COMPLICATIONS**A) During Pregnancy**

1. Abdominal discomfort and backache.
2. Malpresentation.
3. Nonengagement of the presenting part.

B) During Labour

1. Prolonged labour due to misdirection of uterine force and weak abdominal muscles.
2. Premature rupture of membranes and prolapse of cord because of malpresentation and nonengagement of the presenting part.
3. Rupture of uterus due to misdirection of uterine force so the fetus is pushed against the posterior uterine wall.

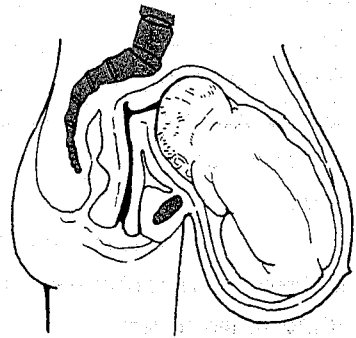


Fig. 41. Pendulous abdomen

MANAGEMENT

A corset is worn during pregnancy. During labour the patient is kept in the dorsal position with application of a tight abdominal binder. Contracted pelvis must be excluded.

GRAND MULTIPARITY

The grand multipara is a woman with 5 or more deliveries. The incidence is 8-12% in Britain and 26.3% in Egypt (Hefnawi). She is liable to the following complications:

A) During Pregnancy

There is increased incidence of:

1. Abortion.
2. Preterm labour.
3. Anaemia (iron deficiency and folic acid deficiency anaemia).
4. Pendulous abdomen.
5. Malpresentation due to lax abdominal and uterine muscles.
6. Nonengagement of presenting part.
7. Pre-eclampsia.
8. Abruptio placentae due to chronic hypertension.
9. Placenta praevia.
10. Diabetes mellitus.

B) During Labour

1. Uterine atony due to increased fibrous and elastic tissues.
2. Postpartum haemorrhage.
3. Placenta accreta as the decidua basalis is absent or poorly formed.
4. Obstructed labour due to malpresentation, large baby and osteomalacic changes in the pelvis.
5. Rupture of uterus due to obstructed labour and degenerative muscle fibres.
6. Increased incidence of operative interference.
7. Higher maternal mortality.

ELDERLY PRIMIGRAVIDA

A woman who is pregnant for the first time and her age is 35 years or above.

COMPLICATIONS**A) During Pregnancy**

There is increased incidence of:

1. Hyperemesis gravidarum.
2. Abortion.
3. Preterm labour.
4. Pre-eclampsia.
5. Abruption placentae due to hypertensive disease.
6. Some diseases as hypertension, diabetes mellitus and heart disease become worse as age advances and this will affect the outcome of pregnancy. So in the presence of such diseases the woman is advised to complete her family before the age of 30.
7. Increased incidence of congenital fetal malformation as Down syndrome.

B) During Labour

1. Abnormal uterine action due to anxiety.
2. Prolonged labour.
3. Signs of maternal distress develop rapidly.
4. The perineum and lower genital tract are rigid so episiotomy is often indicated.

Note: The incidence of Down syndrome (Trisomy 21) increases with maternal age, so it is about 1/2000 at the age of 20, 1/1000 at 30 years, 1/250 at 35 years, and 1/100 at 40 years.

ABDOMINAL PAIN DURING PREGNANCY

The causes of pain may be:

I) Uterine

1. Abortion.
2. Preterm labour and false labour pains.
3. Breech presentation when the head causes discomfort in one hypochondrium.
4. Acute polyhydramnios.
5. Multiple pregnancy.
6. Hydatidiform mole.
7. Abruptio placentae.
8. Rupture of uterus.
9. Fibroid complicated by red degeneration or torsion.
10. Rupture of angular or rudimentary horn pregnancy.
11. Incarcerated retroverted gravid uterus. The distended bladder causes lower abdominal pain.
12. Stretching of round ligaments. This leads to stretching of the nerve fibres and pain along the round ligament and in the groin.
13. Severe fetal movements may cause discomfort.
14. Torsion of pregnant uterus which is very rare.

II) Tubal

Tubal pregnancy and acute salpingitis (very rare during pregnancy). The presence of a pregnancy in the uterus prevents ascending infection. It is, however, possible for pregnancy and gonococcal infection to occur at the same time.

III) Ovarian

1. Complication in an ovarian tumour as torsion.
2. Ovarian pregnancy.
3. Haemorrhage into the corpus luteum of pregnancy.
4. Stretching of the ovarian capsule by the corpus luteum of pregnancy or theca lutein cysts of hydatidiform mole.

IV) Varicocele and haematoma of the broad ligament.

V) Gastrointestinal tract: As appendicitis, gallstone, and affection of the liver in pre-eclampsia.

VI) Urinary tract: As cystitis, pyelonephritis and renal colic. Acute retention of urine occurs with incarcerated retroverted gravid uterus.

VII) Abdominal wall

1. A primigravida may complain of pain around the umbilicus and at the attachment of the rectus muscle with the ribs due to stretching of tissues.
2. Sudden cough or strain may cause laceration of the rectus muscle with haematoma formation.

VIII) Miscellaneous

1. Rupture of the spleen or splenic aneurysm.
2. Acute porphyria presents with abdominal pain, psychiatric disturbance, and autonomic effects. Pregnancy may precipitate an attack and so it may present for the first time during pregnancy.
3. Sick cell crisis in a patient with sickle cell disease.

NORMAL LABOUR

ANATOMY OF THE FEMALE PELVIS

The pelvis is divided into:

1. *False pelvis*: Above the pelvic brim (linea terminalis).
2. *True pelvis*: Below the pelvic brim.

TRUE PELVIS

It has inlet, cavity and outlet.

1) Pelvic Inlet (Pelvic Brim or Superior Pelvic Strait)

Boundaries

The upper border of symphysis pubis, pubic crests, pubic tubercles, upper border of the superior pubic rami, iliopectineal eminences, iliopectineal lines, sacroiliac joints, alae of the sacrum and sacral promontory.

Plane of Inlet

It is an imaginary plane passing by its boundaries. In the standing position the plane of inlet lies obliquely and makes an angle of 55° with the horizon.

Diameters of the Pelvic Inlet

1. Antero-posterior Diameters

- a) *The anatomical antero-posterior diameter or true conjugate*: It extends from the middle of the sacral promontory to the middle of the upper border of the symphysis pubis, about 11 cm.
- b) *The obstetrical antero-posterior diameter or the obstetrical conjugate*: Extends from the middle of the sacral promontory to the most bulging point on the posterior surface of the symphysis pubis. It is the actual diameter which meets the head during its descent, about 10.5 cm.
- c) *The diagonal or oblique conjugate*: From the middle of sacral promontory to the lower border of symphysis pubis, about 12.5 cm. It is 1.5 cm longer than the true conjugate. This difference varies according to the length and inclination of the symphysis pubis.

2. The Transverse Diameters

- a) *The anatomical transverse diameter*, about 13 cm. It is the distance between the two furthest points on the iliopectineal lines. In the normal female pelvis, the anatomical

transverse diameter is nearer to the promontory than to the symphysis pubis. Because this diameter does not pass by the center of the inlet, it is not utilized by the fetal head during its descent.

- b) *The obstetrical or available transverse diameter* is that which passes by the center of pelvic inlet. It is the diameter utilized by the fetal head during its descent.
3. The oblique diameter (about 12 cm) extends from the sacroiliac joint on one side to the iliopectineal eminence on the other side. The right oblique diameter extends from the right sacroiliac joint and vice-versa.
4. The sacrocotyloid diameter (9.5 cm) from the middle of sacral promontory to the iliopectineal eminence of one side. This diameter becomes occupied by the biparietal diameter of the head in occipito-posterior position leading to delay in descent of the head.

N.B.: The sacral promontory is the upper border of the anterior surface of the first sacral vertebra.

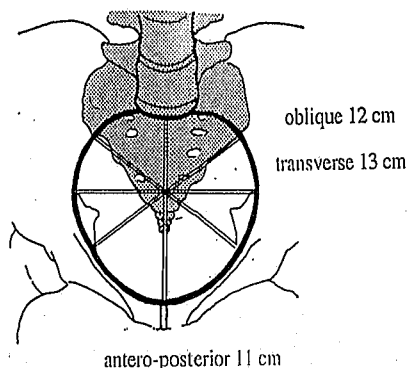


Fig. 42. Diameters of pelvic inlet

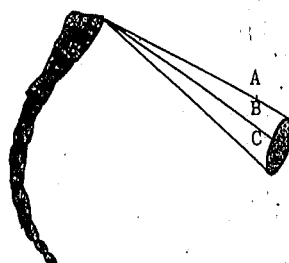


Fig. 43. Conjugate diameters

(A) True, (B) Obstetrical, (C) Diagonal

II) The Pelvic Outlet (Inferior Pelvic Strait)

Diameters of the Pelvic Outlet

1. Antero-posterior Diameters

- a) *Anatomical antero-posterior diameter.* It extends from the lower margin of symphysis pubis to the tip of coccyx, it is 11 cm.
- b) *Obstetrical antero-posterior diameter.* Extends from the lower margin of symphysis pubis to the tip of sacrum. It is 13 cm. During labour the coccyx is pushed backwards by the fetal head to be in line with the sacrum.

2. Transverse Diameters

- a) *Anatomical transverse or intertuberos (bituberos) diameter*: It is the distance between the inner surfaces of the ischial tuberosities, about 11 cm.
- b) *The obstetrical transverse or the interspinous (bispinous) diameter*. It is the distance between the tips of the ischial spines, about 10.5 cm. Less than 9.5 cm indicates contracted midpelvis.

The Anatomical Outlet

Boundaries

The lower margin of symphysis pubis, the combined rami of the ischium and pubis, the ischial tuberosities, sacrotuberous and sacrospinous ligaments and the tip of coccyx.

Plane of the Anatomical Outlet

It is an imaginary plane passing by its boundaries. There are two triangular planes, the anterior triangle or anterior sagittal plane and the posterior triangle or posterior sagittal plane with a common base at the line joining the two ischial tuberosities. The anterior triangle has its apex at the lower margin of symphysis pubis, while the apex of the posterior triangle is at the tip of coccyx.

Thom's Rule

If the sum of the intertuberos and posterior sagittal diameters is less than 15 cm, the pelvic outlet is contracted and caesarean section should be performed to deliver a living fetus.

N.B.: The posterior sagittal diameter (7.5–10 cm) extends from the center of the intertuberos diameter to the tip of sacrum.

The Obstetrical Outlet

Boundaries

It is the lower segment of the pelvis which is bounded below by the anatomical outlet, posteriorly by the coccyx and above by the plane of least pelvic dimensions which passes by the lower border of symphysis pubis, the ischial spines and tip of sacrum.

Plane of Obstetrical Outlet

It is the plane of least pelvic dimensions. Its transverse diameter is the interspinous diameter which is the smallest diameter of the pelvis (10.5 cm).

III) The Pelvic Cavity

It lies between the symphysis pubis and sacrum.

The plane of the pelvic cavity, or the plane of the greatest pelvic dimensions; extends between the middle of the posterior surface of symphysis pubis, and the junction between the second and third sacral vertebrae. This plane has a rounded shape, all its diameters are equal and measure 12 cm. Internal rotation of the head takes place when the biparietal diameter of the head occupies this wide plane of the pelvis.

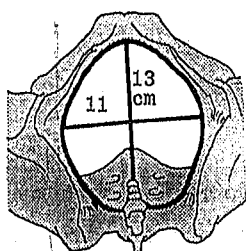


Fig. 44. Anatomical pelvic outlet

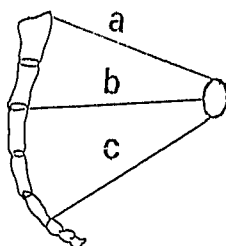


Fig. 45. Planes of the pelvis. (a) Plane of inlet, (b) Plane of greatest pelvic dimensions, (c) Plane of least pelvic dimensions

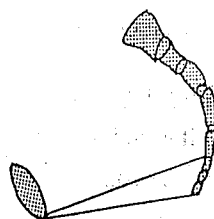


Fig. 46. Obstetric outlet

THE PELVIC AXES

1. The Anatomical Pelvic Axis (Curve of Carus)

It is an imaginary line, passing by the center of the inlet, cavity and outlet of the pelvis. It is "C" shaped and runs parallel with the curve of the sacrum. It has no obstetric value because it is not utilized by the head during its delivery.

2. The Obstetrical Pelvic Axis

It is the direction taken by the head during its delivery. At first the head descends downwards and backwards until it reaches the level of the ischial spines where the head meets the pelvic floor and then it passes downwards and forwards. Therefore the obstetrical axis is J or L-shaped.

Note: The planes of the pelvis are the plane of pelvic inlet, plane of pelvic outlet, plane of greatest pelvic dimensions and plane of least pelvic dimensions.



Fig.47. Anatomical pelvic axis



Fig.48. Obstetrical pelvic axis

THE FETUS

The Fetal Attitude

It is the relation of the fetal parts to each other. In most cases the fetus is in an attitude of general or complete flexion, that is, all joints are flexed.

Lie

It is the relation between the long axis of the fetus and that of the mother. If they are parallel the lie is longitudinal but if they cross each other, the lie is oblique or transverse.

Presentation

It is the first part of the fetus which meets the pelvic brim, and it is the first part felt by vaginal examination. The presentation may be cephalic (96%), breech (3.5%) or shoulder (0.5%).

Denominator

It is a landmark on the presenting part by which the position of the fetus is known. In vertex presentation, the occiput is the denominator.

Position

It is the relation of the denominator to the maternal pelvis.

In vertex presentation there are 8 positions. Direct occipito-anterior (the occiput points towards the symphysis pubis), right occipito-anterior (it points towards the right iliopectineal eminence), right occipito-transverse (it points towards the midpoint of the right iliopectineal line), right occipito-posterior (it points towards the right sacroiliac joint), direct occipito-posterior (it points towards the sacral promontory), left occipito-posterior, left occipito-transverse and left occipito-anterior.

The commonest is the left occipito-transverse. Occipito-anterior positions are commoner than occipito-posterior positions, because of better accommodation between the concavity of the front of the fetus and the convexity of the lumbar spine of the mother. Left occipito-anterior is commoner than right occipito-anterior, because in the former the head descends in the right oblique diameter of the pelvis, while in the latter the head descends in the left oblique diameter of the pelvis, which is reduced by the presence of pelvic colon. Also the right oblique diameter is anatomically slightly longer than the left one due to the more frequent use of the right leg. For the same reason, ROP is commoner than LOP.

N.B.: Cephalic presentation is more common than breech because the head is heavier than the breech and because the breech is larger, it occupies the wide fundus.

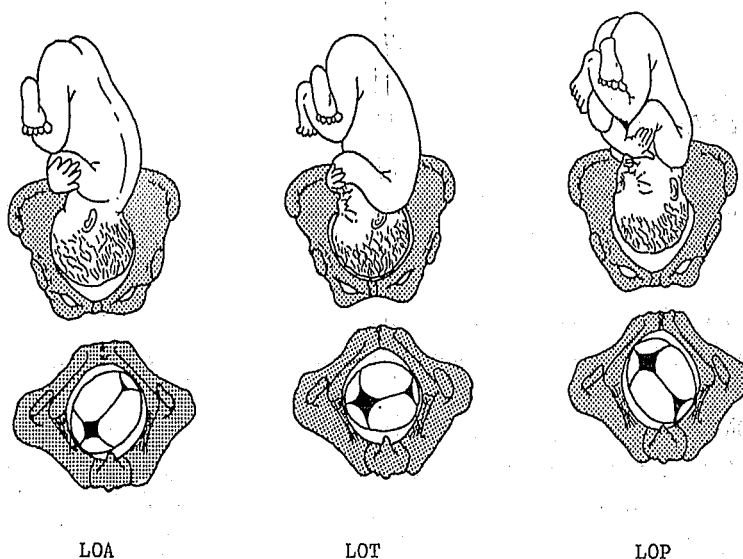


Fig. 49. Vertex presentations

FETAL SKULL

It is divided into vault, face and base. The vault is made of soft flat bones separated by sutures and fontanelles.

Bones of the Vault

Two frontal bones; 2 parietal bones, 2 temporal bones and 1 occipital bone.

Sutures

Spaces between bones, and made of unossified membranes. They are six in number.

- *Frontal suture*; between the two frontal bones.
- *Sagittal suture*; between the two parietal bones.

- *Coronal suture*; between the frontal and parietal bones.
- *Lambdoidal suture*; between the two parietal bones and occipital bone.
- *Two temporal sutures*; each between the parietal and temporal bone.

Fontanelles

Areas where sutures meet. They are six in number.

Anterior Fontanelle or Bregma

It is large, lozenge shaped, formed by the meeting of four bones, two frontal and two parietal bones, has a soft membranous floor, becomes obliterated 1.5 years after birth.

Posterior Fontanelle or Lambda

Small, triangular in shape, formed by the meeting of three bones, two parietal bones and the occipital bone, has a bony floor, it is obliterated at full term, membranous in the preterm baby.

The anterior temporal fontanelles are at the junction of the temporal and coronal sutures. The posterior temporal fontanelles are at the junction of the temporal and lambdoidal sutures.

Vertex

It is the area of the vault of the skull between the anterior and posterior fontanelles and the parietal eminences.

Diameters of the Fetal Head

A) Transverse Diameters

1. Biparietal (9.5 cm); between the two parietal eminences.
2. Subparietal-supraparietal (9 cm), from below one parietal eminence to above the other.
It is 0.5 cm less than the biparietal diameter.
3. Bitemporal (8 cm), between the anterior ends of temporal sutures.
4. Bimastoid (7.5 cm), between the tips of the mastoid processes.

B) Longitudinal Diameters or Engaging Diameters

The engaging diameter is the longitudinal diameter of the presenting part.

1. The suboccipito-bregmatic diameter (9.5 cm), measured from the suboccipital point, i.e. the junction of the head and neck posteriorly to the center of the anterior fontanelle. It is the diameter of engagement in occipito-anterior position when the head is completely flexed.

2. The suboccipito-frontal diameter (10 cm), measured from the suboccipital point to the anterior end of anterior fontanelle. It is the diameter of engagement in occipito-anterior position when the head is not completely flexed.
3. The occipito-frontal diameter (11.5 cm), measured from the occipital protuberance to the root of the nose. It is the diameter of engagement, in occipito-posterior position.
4. The mento-vertical diameter (13.75 cm), measured from the tip of the chin to the vertical point which is the point at the middle of sagittal suture. It is the diameter of engagement in brow presentation.
5. The submento-bregmatic diameter (9.5 cm), measured from the junction of chin and neck to the center of the anterior fontanelle. It is the engaging diameter in face presentation when the head is completely extended.
6. The submento-vertical diameter (11.5 cm), measured from the junction of the chin and neck to the vertical point. It is the diameter of engagement in face presentation when the head is not completely extended. It is also the diameter which distends the vulva during delivery of face presentation.

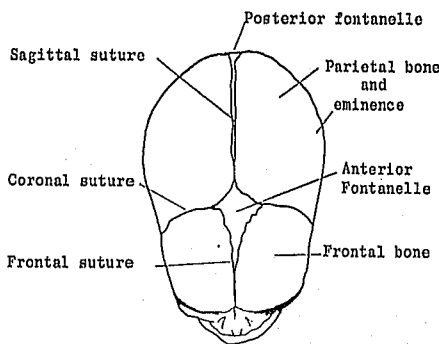


Fig. 50. Fetal skull

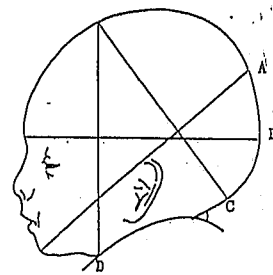


Fig. 51. Diameters of fetal head

(A) Mento-vertical, (B) Occipito-frontal
(C) Suboccipito-bregmatic, (D) Submento-bregmatic

Caput Succedaneum

It is oedema of the fetal scalp due to prolonged compression of the fetal head against the maternal tissues, leading to interference with the venous return. It occurs in obstructed labour after rupture of membranes. The caput is few millimeters in thickness, but it may be large and protrudes from the vagina giving a wrong impression that the head is low, when actually it is not engaged. The caput disappears within one or two days after delivery. The presence of the caput indicates that: (1) the fetus was alive during labour, (2) labour was prolonged and obstructed; (3) the site of the caput gives idea about the position of the head during labour because it is formed over the lowest part of the head.

Note: Caput means head and succedaneum means swelling.

Moulding

It means that the flat bones of the fetal skull overlap each other, one parietal bone overlaps the other and both parietals overlap the occipital bone. In severe degrees, the parietal bones overlap the frontal bones. Slight moulding is physiological and beneficial because it diminishes the diameters of the head and facilitates its passage through the birth canal. Severe or rapid moulding is dangerous as it may lead to intracranial haemorrhage.

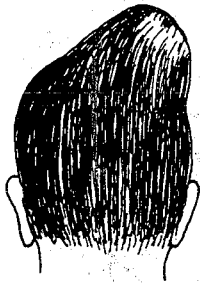


Fig. 52. Caput succedaneum

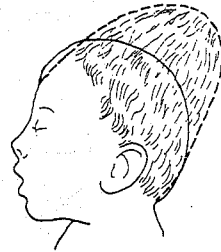


Fig. 53. Moulding

Synclitism

This means that the head descends with the two parietal bones at the same level so the sagittal suture lies midway between the promontory and symphysis pubis. It occurs in 25% of labours.

Asynclitism

The head descends with one parietal bone at a lower level than the other due to lateral flexion of the head on the shoulders. Asynclitism is common and occurs in 75% of labours.

It has a mechanical advantage, because the head will enter the pelvis by a smaller diameter which is the subparietal-supraparietal (9 cm) instead of the biparietal diameter (9.5 cm.).

There are two types:

- a) *Anterior asynclitism* (anterior parietal presentation or Nägele obliquity): The anterior parietal bone is at a lower level than the posterior, and the sagittal suture is nearer to the promontory than to the symphysis pubis. It is more common in multipara with lax abdominal wall allowing the uterus to fall forwards.
- b) *Posterior asynclitism* (posterior parietal presentation or Litzmann obliquity): The posterior parietal bone is at a lower level than the anterior, the sagittal suture is nearer to the symphysis pubis than to the promontory. It is more common in primigravidae.

because the strong abdominal muscles push the fetus backwards against the maternal spine.

N.B.:

- Anterior parietal presentation is better because during correction of asynclitism the head will meet resistance at the sacral promontory which is a thin edge while in posterior parietal presentation the head will meet resistance along the whole length of symphysis pubis.
- Some consider anterior asynclitism as posterior parietal presentation and vice-versa.

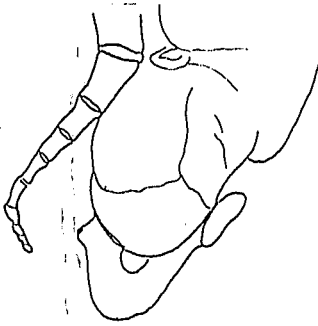


Fig. 54. Anterior parietal presentation

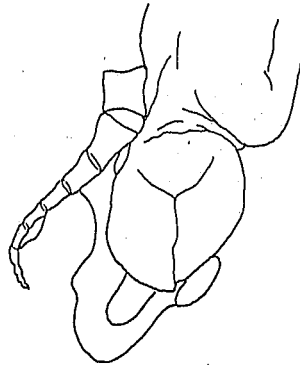


Fig. 55. Posterior parietal presentation

Engagement

Engagement of the head means that the biparietal diameter of the fetal head, i.e. largest transverse diameter, has passed below the pelvic brim, so by abdominal examination the head is not felt and by vaginal examination the vertex is at or below the level of the ischial spines. In primigravida the head should engage at 38 weeks of pregnancy due to strong abdominal and uterine muscles. In multipara because these muscles are lax, the head may not engage until labour begins or even when the second stage starts. Engagement of the breech means the passage of the bitrochanteric diameter below the pelvic brim.

Causes of Non-Engagement of the Head in the Primigravida during the Last 2 Weeks of Pregnancy

A) Fetal Causes

1. Large head.
2. Hydrocephalus.
3. Malposition of the head as occipito-posterior, face and brow.
4. Multiple pregnancy.
5. Short umbilical cord.
6. Polyhydramnios.

B) Maternal Causes

1. Pelvic contraction.
2. Pelvic tumour.
3. Placenta praevia.
4. Full bladder or rectum.
5. Hypertonic lower uterine segment.
6. Atony of abdominal muscles.
7. High assimilation pelvis, i.e. 6 sacral vertebrae.
8. Wrong calculation.

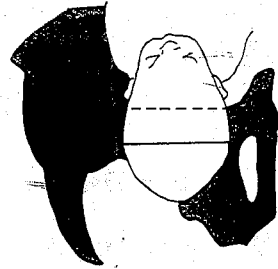


Fig. 56. Engagement of head

C) Idiopathic

In 20% of cases there is no apparent cause.

However, the common cause is occipito-posterior position.

PELVIMETRY

It is estimation of pelvic diameters.

METHODS

1. Clinical Pelvimetry.
2. Radiological Pelvimetry.
3. Pelvimetry using computed tomography (CT scanning).
4. Pelvimetry using magnetic resonance imaging (MRI).
5. Tests for cephalopelvic disproportion.

**Clinical Pelvimetry**

This includes inlet, outlet and internal pelvimetry.

Inlet Pelvimetry

1. *Interspinous diameter* (25 cm). It is the distance between the anterior superior iliac spines.
2. *Intercristal diameter* (27.5 cm). The distance between the two furthest points on the outer borders of the iliac crests.
3. *External conjugate* (20 cm). Between the upper border of symphysis pubis and a point on the back which is opposite the sacral promontory. This point is immediately below the tip of the last lumbar spine. If we subtract 9 cm we know the length of the true conjugate diameter (11 cm).

These are diameters of the false pelvis and have no obstetric value.

Outlet Pelvimetry

1. *The obstetrical antero-posterior diameter* (13 cm) from the lower border of symphysis pubis to the tip of sacrum. Subtract 1 cm for the thickness of sacrum.
2. *The intertuberous diameter* (11 cm). Between the two ischial tuberosities and add 1 cm for the thickness of fat. It is roughly estimated by trying to insert the closed fist of the hand.
3. *Thom's rule*. See page (125).

Internal Pelvimetry: See contracted pelvis.

Radiological Pelvimetry: See contracted pelvis.

Pelvimetry using Computed Tomography: See contracted pelvis.

Pelvimetry using Magnetic Resonance Imaging: See contracted pelvis.

Tests for Cephalopelvic Disproportion: See contracted pelvis.

NORMAL LABOUR

- **Normal labour** means a mature fetus presenting by the vertex is delivered spontaneously through the birth canal within 24 hours without interference except episiotomy and without fetal or maternal complication.
- **Preterm or premature labour** (curtailed pregnancy): It is labour occurring after viability of the fetus (20 weeks) and before 38 weeks of pregnancy (37 completed weeks).
- **Mature or term infant**: It is an infant born between 38 and before 42 weeks of pregnancy.
- **Postterm pregnancy** (postmaturity): Prolongation of pregnancy two weeks or more beyond the expected date of delivery, i.e., the duration of pregnancy is 42 weeks or more (294 days or more).

THE CAUSE OF ONSET OF LABOUR

Not definitely known. There are several theories but none of them is completely satisfactory. An example is the fetal cortisol theory. Before onset of labour there is increased production of cortisol by the fetal adrenal glands. Cortisol leads to decreased formation of progesterone in the placenta and increased production of oestrogen and prostaglandins. The latter will stimulate uterine contractions leading to labour pains. In case of anencephaly with small adrenal glands pregnancy may be prolonged. This theory is proven in the sheep but not in human.

CALCULATION OF THE EXPECTED DATE OF DELIVERY (EDD)

A) Symptoms

1. *Duration of Amenorrhoea*: The duration of pregnancy is 280 days, or forty weeks, calculated from the first day of the last normal menstruation. So we add seven days to the first day of the last menstruation and count forwards 9 calendar months (Näegele rule). The commonest method used in practice, correct in most cases, within one week on either side. This menstruation-labour interval (280 days) is not the true duration of pregnancy which is the fertilization-labour interval (266 days). Only 4% of women will deliver on the expected date.
2. *Date of Single Coitus* (as in case of rape): Date of single coitus + 9 calendar months -7 days.
3. *Date of Quickening*: Add 22 weeks in the multigravida and 20 weeks in the primigravida.
4. *Lightening*.

B) Signs

1. *Fundal level*. See page 11.
2. *The height of the fundus* above the symphysis pubis in centimeters gives the duration of pregnancy in weeks. This rule is applied only between 20 and 36 weeks of gestation (McDonald measurement).
3. *Engagement of the head*: In most primigravidae and in many multigravidae the head engages in the pelvis at 38 weeks of pregnancy..
4. *Shelfing*.
5. *Sonography*: Gestational age is determined by measuring the biparietal diameter, head circumference, abdominal circumference, femur length and in the first trimester by crown-rump length between 6 and 12 weeks, and before that by measuring the diameter of the gestational sac.
6. *Radiological Examination*
 - a) Length of biparietal diameter: 7.5 cm at 32 weeks, 8.5 cm at 36 weeks, 9.5 cm at 40 weeks.
 - b) Ossification centers: Lower end of femur at 36 weeks, upper end of tibia at 40 weeks.

X-ray examination has been replaced by ultrasound.

Lightening

In most primigravidae and in many multigravidae the fetal head engages in the pelvis at 38 weeks of pregnancy. This leads to relief of dyspnoea and palpitation and the patient

starts to complain of frequency of micturition, heaviness in the pelvis and difficulty in walking. In multipara the head may remain above the pelvic brim and only engages in the pelvis after the onset of labour and even after the start of the second stage. In the primigravida because of strong uterine and abdominal muscles, engagement of the head occurs at 38 weeks of pregnancy. If this fails to occur the cause must be looked for (page 132).

Shelfing

The fundus of the uterus falls forwards when the fetus becomes perpendicular to the pelvic inlet after engagement of the head.

Differentiation between Pregnancy at 32 and 40 Weeks

1. *Symptoms:* Duration of amenorrhoea, date of quickening and lightening.
2. *Signs:* Shelfing of the fundus, size of the fetus, engagement of the head, ultrasound or radiological examination.

Note: The accuracy of ultrasound in measuring gestational age is up to ± 1 week in the first trimester, ± 2 weeks in the second trimester and ± 3 weeks in the third trimester.

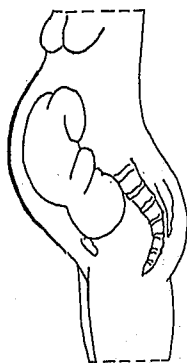


Fig. 57. Shape of abdomen before lightening

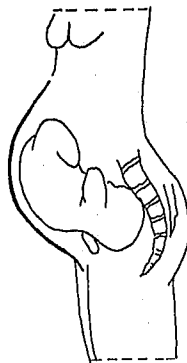


Fig. 58. Shape of abdomen after lightening (shelfing)

DIAGNOSIS OF ONSET OF LABOUR

- *Symptoms:* (1) True labour pains, (2) the passage of the show.
- *Signs:* (1) Dilatation of the internal cervical os, (2) formation of the bag of forewaters.

True Labour Pains

During pregnancy there are painless intermittent uterine contractions which are felt on palpating the uterus. These are known as Palmer sign in early pregnancy and as Braxton-Hicks contractions in late pregnancy. True labour contractions are: (1) painful causing

colicky pain in the lower abdomen and backache (cervical dilatation); (2) they are regular and increase gradually in strength, duration and frequency; (3) they are effective, i.e. cause dilatation of the internal os; (4) they are accompanied by hardening of the uterus; (5) the bag of forewaters becomes tense during contraction; (6) they are usually increased by an enema because a full rectum reflexly inhibits uterine contractions.

The Show

It is a blood stained mucous discharge noticed at the start of labour. The mucus is the cervical mucous plug which normally fills the cervical canal and is expelled when the cervix starts to dilate. The blood is caused by separation of the membranes from the lower uterine segment or minute lacerations of cervical mucosa. Labour usually starts within 24 hours after the passage of the show.

Dilatation of the Internal Cervical Os

A closed internal os means labour has not started, however, the external os or even the internal os may admit 1 or 2 fingers before the onset of labour, especially in a multigravida.

Formation of the Bag of Forewaters

The lower pole of the fetal membranes (chorion and amnion), separates from the lower uterine segment to form a bag of water which bulges through the cervix and becomes tense during uterine contraction (a sure sign).

False Labour Pains

Sometimes the intermittent uterine contractions of pregnancy cause some degree of abdominal pain for several days or weeks before the onset of true labour pains. These false pains are irregular, do not increase in strength, frequency or duration. The bag of forewaters does not bulge during the contraction. They are usually relieved by sedatives or enema.

STAGES OF LABOUR

1. First Stage (Stage of Cervical Dilatation)

Starts with the onset of true labour pains and ends when the cervix is fully dilated. It takes about 12 hours in the primigravida and 6 hours in the multigravida.

2. Second Stage (Stage of Expulsion of the Fetus)

Begins when the cervix is fully dilated and ends with delivery of the fetus. It takes about one hour in the primigravida and half an hour in the multigravida.

3. Third Stage (Stage of Expulsion of the After-Birth)

Starts with delivery of the fetus and ends with expulsion of the placenta, cord and membranes, takes about 10-30 minutes (average 10 minutes) and is painless.

Note: Labour is prolonged when it lasts more than 18 hours.

Criteria of the Second Stage of Labour

1. The patient starts to bear down during uterine contractions due to pressure of the presenting part on the rectum and pelvic floor causing reflex involuntary contraction of the diaphragm and abdominal muscles.
2. The patient feels the desire to evacuate the rectum or bladder.
3. Rupture of the membranes and flow of the amniotic fluid. Normally this occurs when the cervix is fully dilated, however, it may occur early in labour in the first stage or even before the start of labour (premature rupture of membranes). It may not occur at all and the fetus is delivered inside the intact amniotic sac.
4. Full dilatation of the cervix: When it is fully dilated it is 4 inches or 10 cm. in diameter and can admit 5 fingers (this is the sure sign).

Note:

Active and Latent Phases of the First Stage of Labour: Normally the cervix dilates slowly to reach 3 cm in the nullipara and 4 cm in the multipara (latent phase) and then rapidly to reach 9 cm (active phase) and then slowly to full dilatation (sigmoid curve). A latent phase is considered prolonged if it lasts more than 20 hours in the primigravida and more than 14 hours in the multigravida. During the active phase the primigravida usually dilates 1.2 cm per hour and a multiparous patient dilates 1.5 cm per hour.

THE MECHANISM OF NORMAL LABOUR

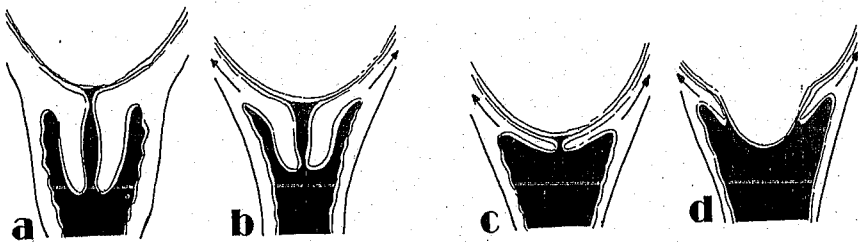


Fig. 59. Cervical dilatation in primigravida. The cervix is taken up then external Os dilates.

1) Cervical Dilatation

It is due to contraction and retraction of the uterus which push the head or bag of forewaters through the cervix. In primigravida the cervix dilates from above downwards.

The internal os dilates at first, then the cervical canal is taken up or effaced and finally the external os starts to dilate because its muscle fibres are still intact and resist dilatation. In multigravida, dilatation of the internal os, taking up of the cervix and dilatation of the external os occur together at the same time.

Note: Cervical effacement means the portio vaginalis of the cervix becomes short and thin. Before labour starts the portio vaginalis of the cervix is about 2 cm long and is said to be uneffaced or 0% effaced. When it is 1 cm long it is 50% effaced and when it is 0.5 cm it is 75% effaced and when less than 0.25 cm it is 100% effaced.

II) Expulsion of the Fetus

A. *Delivery of the Head*

The following movements occur in the head during its delivery:

1. Descent.
2. Engagement.
3. Increased flexion.
4. Internal rotation.
5. Extension.
6. Restitution.
7. External rotation.

1. Descent

It is a constant movement throughout delivery. It is caused by:

- a) Uterine contraction and retraction.
- b) Contraction of the diaphragm and abdominal muscles when the patient starts to bear down.
- c) Unfolding of the fetus caused by contraction of circular muscle fibres of the uterus.

2. Engagement

It is the passage of the biparietal diameter of the head below the pelvic brim.

3. Increased Flexion

As the head descends it meets resistance from the pelvic walls and pelvic floor, and this leads to increased flexion of the head. Flexion is explained by the two-armed lever theory. (The head acts as a lever with a fulcrum at the atlanto-occipital joint. The short arm of the lever extends from the occiput to the joint and the long arm extends from this joint to the sinciput. When the head meets resistance during its descent the force applied to the long arm is greater than that applied to the short arm. This leads to ascent of the sinciput and descent of the occiput causing complete flexion).

4. Internal Rotation

As a result of increased flexion the occiput meets the pelvic floor first and so it rotates forwards. In occipito-anterior position, the occiput rotates forwards one-eighth of a circle, while in occipito-posterior it rotates forwards three eighths of a circle so that the occiput becomes behind the symphysis pubis. Internal rotation is due to several factors:

- The sloping character of the pelvic floor. This is the main factor. The pelvic floor is made by the two levator ani muscles. The muscle passes downwards, forwards and medially to meet its fellow of the opposite side. When the occiput meets the pelvic floor, it will move in the direction of the levator ani that is downwards, forwards and medially.
- The shape of the pelvis, the pelvic inlet is a transverse oval, while the pelvic outlet is a longitudinal oval, and this will act in a screw action.
- Unequal flexibility of the different parts of the fetus as proved by experiments on models carried out by Selheim.

5. Extension

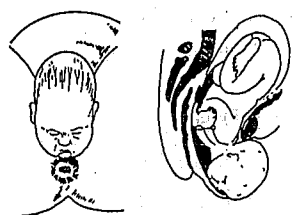
After internal rotation has been completed the head continues to descend until the suboccipital region appears below the symphysis pubis. Now the head is delivered by extension, the vertex, the forehead, the face and lastly the chin comes out successively. Extension is due to contraction of pelvic floor pushing the head upwards and forwards.

6. Restitution

After delivery of the head, the occiput rotates one-eighth of a circle in a direction opposite to that of internal rotation to undo the twist of the neck caused by internal rotation.

7. External Rotation

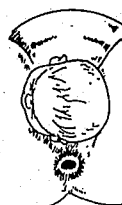
The occiput undergoes another external rotation one-eighth of a circle in the same direction as that of restitution. This is due to internal rotation of the anterior shoulder one-eighth of a circle. This movement is transmitted to the delivered head. So at the end the occiput is towards one thigh and the face is towards the other thigh.



Delivery of head by extension



Restitution



External rotation

Fig. 60. Delivery of head

B. Delivery of the Shoulders and Body

The biacromial diameter descends in the opposite oblique diameter of the pelvis. The anterior shoulder meets the pelvic floor first and rotates forwards one-eighth of a circle. With further descent, the anterior shoulder appears below the symphysis pubis. The posterior shoulder is delivered first by lateral flexion of the fetal spine then the anterior shoulder follows. Finally the trunk is delivered by lateral flexion of the spine around the symphysis pubis.

III) Mechanism of the Third Stage of Labour

After delivery of the fetus the uterine contractions continue in the third stage with the same frequency and strength as those of the second stage, however, they are painless contractions. As a result of uterine retraction the placental area diminishes in size from about 20 cm to 7 cm. Because the placenta is inelastic and unable to shrink it starts to separate from the uterine wall. There are two mechanisms for separation of the placenta; the Schultze and Duncan mechanism. The separated placenta is expelled by the bearing down efforts of the patient.

Schultze Mechanism

Commonest, occurs in 80% of cases. The central part of the placenta separates first as the placenta is firmly attached at its edges. A retroplacental haematoma forms and the placenta is delivered by its fetal surface followed by the membranes containing the blood clot. This mechanism is less liable to be followed by bleeding or retained fragments. Actually the placenta is delivered like inverted umbrella.

Duncan Mechanism

In 20% of cases. The placenta separates at its lower edge, and is delivered sideways with the maternal and fetal surfaces appearing together. There is no retroplacental haematoma. It is more liable to be followed by bleeding and retained fragments.

N.B.:

- Contraction means temporary shortening of the muscle fibre.
- Retraction means permanent shortening of the muscle fibre.

Functions of Retraction

1. Dilatation of cervix.
2. Expulsion of fetus.
3. Separation of the placenta.
4. Control of uterine bleeding.
5. Involution of uterus.

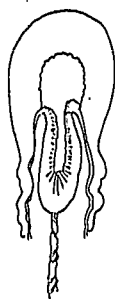


Fig. 61. Schultz mechanism:



Fig. 62. Duncan mechanism



Fig. 63. Shape of uterus after third stage

MANAGEMENT OF NORMAL LABOUR

When the patient is seen in labour, a history is taken. General, abdominal and vaginal examinations are carried out and fetal heart is monitored. Urine is examined for protein and sugar.

1) Management of the First Stage

1. The vulva is shaved and washed from before backwards with an antiseptic solution as Savlon. This is repeated several times during labour. The vulva is kept covered by a sterile pad.
2. The patient is allowed to walk about. The weight of the head and bag of forewaters help in mechanical dilatation of the cervix, also pressure on the lower uterine segment and cervix reflexly stimulates the uterus to contract "reflex polarity". In lower animals and may be in human, pressure on the cervix causes release of oxytocin from the posterior pituitary gland "Ferguson reflex". Rest in bed is recommended when the membranes are ruptured. When the patient lies in bed she is advised to lie on her side to avoid supine hypotensive syndrome (page 89).
3. The bladder is kept empty by frequent micturition every 2 hours or by a catheter passed every four hours if necessary. A distended bladder leads to obstructed labour and uterine atony.
4. The rectum is evacuated by an enema, to avoid uterine atony from a full rectum and to avoid soiling of the vulva and perineum by faeces during delivery of the head.
5. Diet: Nothing is allowed by mouth except water because if vomiting occurs as when anaesthesia is given the vomitus may be aspirated leading to respiratory complications as Mendelson syndrome. Glucose-saline solution is given intravenously (125 ml per hour) if labour is prolonged more than 8 hours.
6. Analgesics are given to relieve pain, e.g. pethidine.

7. The patient is not allowed to bear down during the first stage as this predisposes to genital prolapse, premature rupture of membranes and exhausts the patient.
8. Observation for:
 - a) Maternal pulse, temperature and blood pressure every 2 hours. Blood pressure is recorded every half an hour if it is elevated or below normal.
 - b) Fetal heart sounds at least every 30 minutes or more frequent to detect fetal distress. They are auscultated immediately after the end of uterine contraction. Continuous electronic monitoring is indicated in high risk pregnancy as pre-eclampsia. However, some obstetricians advise continuous monitor for all patients.
 - c) Uterine contractions for frequency, strength and duration. Detected by manual palpation or by external tocometer or by the use of an intrauterine pressure catheter.
 - d) Time of rupture of membranes.
 - e) Degree of cervical dilatation which is assessed by vaginal examination every 2-3 hours.

These observations are recorded in a graphic form, the partogram.

II) Management of the Second Stage

1. When the membranes rupture, vaginal examination is done to estimate the degree of cervical dilatation, to feel the presenting part and to exclude prolapse of umbilical cord.
2. The patient is transferred to delivery room, put in lithotomy or dorsal or left lateral position, vulva and perineum are washed with an antiseptic solution and sterile leggings and towels are applied.
3. The patient is asked to bear down during uterine contractions and to relax in between.
4. Inhalation analgesia as nitrous oxide and oxygen may be required.
5. More frequent observation of the mother and fetal heart sounds (every 5 minutes).
6. The main aim of the second stage of labour is prevention of perineal laceration during expulsion of the head, and this is obtained by:
 - a) When the head appears at the vulva, the perineum is supported during uterine contraction by a sterile pad. This will keep the head flexed until crowning occurs.
 - b) After crowning, the head is allowed to be delivered slowly in the interval between uterine contractions by sliding the perineum over the face. The patient should not bear down during delivery of the head.
 - c) Episiotomy is performed under local anaesthesia when the perineum is over-stretched and going to tear.
7. Once the head is delivered the mouth and nose are cleaned with a piece of gauze to remove mucus or use electric suction. Inspect the neck, if the cord is coiled around it,

try to slip it over the head. If this fails or if there is more than one loop the cord is cut between two clamps.

8. Delivery of the shoulders. The head is grasped between the two hands and traction is applied downwards and backwards until the anterior shoulder appears below the symphysis pubis, then the head is elevated upwards to deliver the posterior shoulder. The trunk is then delivered by gentle traction.
9. The cord is massaged towards the fetus several times. This brings to it 60-90 ml of blood from the placenta. When the pulsations of the cord cease, it is cut between two clamps and the fetus is handed to the assistant. Milking of the cord is contraindicated in erythroblastosis fetalis to avoid further passage of antibodies from the placenta to the fetus.

N.B.: *Crowning* means that the biparietal diameter of the fetal head has passed just outside the vulval ring and the suboccipital region is fixed below the symphysis pubis. When the head is crowned it does not go up in the interval between contractions (before crowning the head descends during uterine contraction, and then recedes up again during uterine relaxation). If the head is allowed to extend before crowning the occipito-frontal diameter (11.5 cm) will pass through the vulva and this predisposes to perineal tear. When the head extends after crowning the vulva will be distended by the suboccipito-bregmatic diameter which is 9.5 cm. For this reason we must support the perineum during each uterine contraction to keep the head flexed until crowning occurs.

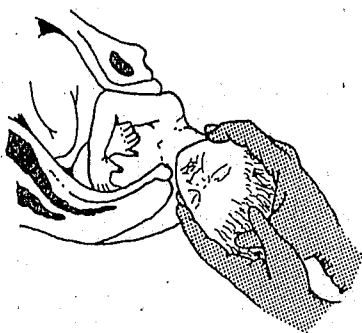


Fig. 64. Delivery of anterior shoulder

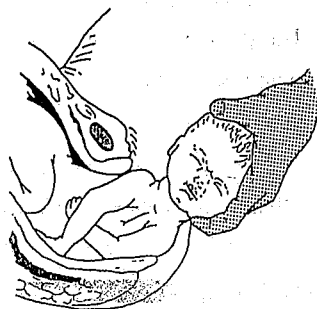


Fig. 65. Delivery of posterior shoulder

III) Management of the Third Stage

1. The placenta is delivered by the conservative or active method.

i) *The Conservative Method*

After delivery of the fetus the left hand is placed over the fundus of the uterus to detect signs of separation and descent of the placenta. When these signs appear the uterus is massaged to contract and then the patient is asked to bear down to deliver the

placenta spontaneously or the contracted uterus is pushed downwards like a piston to expel the placenta (Dublin method). This can be helped by gentle traction on the cord. Ergometrine 0.5 mg is given I.M. or I.V. after delivery of the placenta to diminish bleeding. Also oxytocin can be given by the intravenous drip method. This is the most common method used in the United States.

Signs of Separation and Descent of the Placenta

- a) The body of the uterus becomes smaller, harder and more globular. This is the earliest sign.
- b) The fundus rises upwards because the lower uterine segment becomes distended by the placenta.
- c) A suprapubic bulge may appear due to the presence of the placenta in the lower uterine segment.
- d) Elongation of the cord outside the vulva.
- e) A gush of blood usually occurs from the vagina.

ii) The Active Method

With delivery of the anterior shoulder or even with crowning of the head, ergometrine 0.5 mg is given I.V. or Syntometrine is given I.M. When the fetus is born, the placenta is delivered by the Brandt-Andrews method (see retained placenta). The advantage of the active method is to avoid postpartum haemorrhage because the risk of bleeding is related directly to the time interval between delivery of the fetus and the placenta. However, ergometrine or Syntometrine may cause contraction ring and retention of the placenta.

2. When the placenta appears at the vulva it is rolled between the hands to make a rope of the membranes so that no part of the membranes is missed. The placenta, cord and membranes are inspected. If a placental fragment is missing the uterus is explored under general anaesthesia to remove the retained portion. The cut end of the cord is examined for the presence of 2 arteries and a vein. The absence of one umbilical artery suggests a congenital anomaly in the newborn (25–50%).
3. The perineum, vulva and vagina are examined. Any tear whatever small is sutured immediately or maximally within 24 hours, after that time it is considered as a septic wound. The patient is observed for at least one hour after delivery to detect any postpartum haemorrhage (4th stage of labour).

N.B.: Syntometrine is 0.5 mg ergometrine and 5 units of Syntocinon. I.M. Syntocinon acts after 2 minutes and action lasts 30 minutes, while I.M. ergometrine acts after 4 minutes and action lasts 4 hours. So the drug combines the rapid action of Syntocinon and the prolonged action of ergometrine.

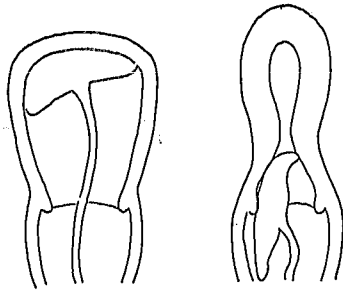


Fig. 66. Signs of descent of the placenta. After separation the body of the uterus becomes smaller and more rounded and the fundus rises upward.

IV) Care of the Newborn

1. Suction of the nose, mouth, and pharynx using a suction catheter (a mucus catheter was used in the olden days).
2. Apgar score is determined 1 and 5 minutes after delivery of the infant (see fetal asphyxia).
3. A plastic umbilical cord clamp is applied 5 cm from fetal abdomen to avoid including an umbilical hernia. The cord is divided distal to the clamp, inspected for bleeding, and painted with alcohol. An abdominal binder is applied. The cord usually falls on the 6th day. If the cord is thick and the umbilical cord clamp cannot be applied, the cord is ligated by 2 thick silk ligatures. The ClampCut can be used. It cuts and clamps the cord at the same time.

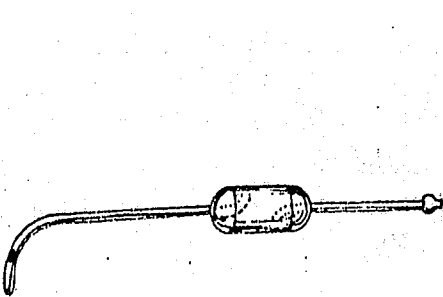


Fig. 67. Mucus catheter

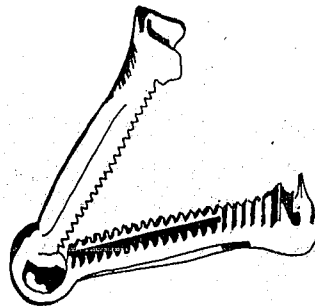


Fig. 68. Hollister umbilical cord clamp.

4. The infant is examined for injuries and congenital anomalies as imperforate anus.
5. Vernix caseosa is removed from the neck, axillae & groins and a bath is not necessary.
6. The infant is weighed, gestational age determined, dressed and a bracelet is applied for identification.

7. Penicillin eye drops or ointment is used to protect against gonococcal ophthalmia neonatorum. Tetracycline or erythromycin ointment can be used.
8. Vitamin K₁ is given to all infants immediately after delivery as 1 mg IM or it is given orally as 1 mg at birth and the dose is repeated at the end of the first, and third weeks (3 oral doses). In high risk infants who are liable to develop haemorrhage as preterm and asphyxiated infants the first dose is given IM and then 2 oral doses are given as above.

ACTIVE MANAGEMENT OF LABOUR

The aim of active management of labour is to ensure that the primigravida will deliver a healthy baby in less than 12 hours. Prolonged labour is more common in primigravidae because of inefficient uterine contractions. Labour is rarely prolonged in multigravidae because the uterus contracts more effectively.

Benefits of Active Management of Labour

It avoids prolonged labour which can lead to: (1) Maternal distress and emotional upset; (2) fetal hypoxia and distress; (3) exhaustion of the medical and nursing staff.

Conditions Needed for Active Management of Labour

(1) A primigravida; (2) single fetus; (3) vertex presentation; (4) at term; (5) absence of fetal distress.

The Principles of Active Management of Labour

1. Antenatal Education

The mother is informed about the physiology of labour and assured that labour will take less than 12 hours. In this way, she can cope better with the stress of labour.

2. Regular Follow-Up of the Patient During Labour

Vaginal examination is done when the patient is admitted to the labour room. This will be repeated every 1–2 hours to know the rate of cervical dilatation which is recorded on the partogram. Dilatation of the cervix follows a sigmoid curve (Friedman). The cervix dilates slowly to reach 3–4 cm (latent phase) then rapidly to reach 9 cm (active phase) and then slowly to full dilatation. The latent phase is about 8 hours in primigravida and 4 hours in multigravidae. The active phase is about 6 hours in primigravida and 2 hours in multigravidae.

3. Correction of Abnormal Progress

The rate of cervical dilatation should not be less than 1 cm per hour in the active phase of labour. If the cervix is not dilating properly, labour is accelerated by rupture of

membranes. Rupture of membranes stimulates uterine contractions and reveals the amount and colour of amniotic fluid. However, if labour is not accelerated within one hour after rupture of membranes, oxytocin infusion is started and gradually increased in strength. Before acceleration of labour, we must exclude cephalopelvic disproportion and fetal distress.

4. Personal Attention

There should be one nurse face to face for each patient throughout labour.

5. Diet

Nothing is allowed by mouth except water. If labour is prolonged more than 8 hours, concentrated glucose is given intravenously at a rate of 125 ml per hour.

6. Provision of suitable analgesia.

7. If labour exceeds 12 hours, caesarean section is considered.

Note: If the presenting part is at the level of ischial spines, it is said to be at "zero station", if above the spines, the distances are stated in minus figures (-1 cm, -2 cm, -3 cm, and "floating"), if below the spines, the distances are stated in plus figures (+1 cm, +2 cm, +3 cm, and "on the perineum"). So if the presenting part is 2 cm above the spines, it is at -2 station.

THE PARTOGRAM

It is a graphic recording of the progress of labour. It includes the following items:

1. General information: Name of the patient, age, diagnosis, time and date of admission, and hospital number.
2. Duration of labour: It is recorded every hour.
3. Fetal heart rate: It is recorded every half an hour.
4. Amniotic fluid: If membranes are intact record (I). If membranes are ruptured, record if spontaneous or artificially ruptured. If the fluid is clear, record (C), if meconium stained, record (M), and if blood stained, record (B).
5. Cervical dilatation is recorded in centimeters.
6. Descent and station of the head: The descent of the head is measured as fifths of the head above the brim. If the head is completely above the brim, it is $\frac{5}{5}$, if 4 fifths are felt, it is $\frac{4}{5}$, and so on. If no part is felt abdominally, it is $\frac{0}{5}$.
The station of the head in relation to the ischial spines is recorded as centimeters above (-) or below (+) the ischial spines.
7. Uterine contractions are recorded and if they are strong or weak and their frequency.
8. Maternal pulse, temperature, and blood pressure are recorded.

9. Urine: The time when urine is passed, its volume, and tests are made for glucose, protein and ketone bodies.
10. Drugs and intravenous fluids: The dose, and route of administration of drugs given during labour as well as amounts of IV fluids are recorded.
11. Fetal blood pH: If done, it is recorded in the space provided.

METHODS OF ASSESSMENT OF FETAL STATUS DURING LABOUR

1. Recording of fetal heart rate (FHR), intermittently by fetoscope or Sonicaid or by continuous electronic fetal monitoring.
2. Sampling of blood from fetal scalp for pH.
3. Observation for the presence of meconium in the amniotic fluid.

RECORDING OF FETAL HEART RATE

The FHR can be continuously recorded by:

1. *The external monitor.* The ultrasound probe (transducer) is applied to the mother's abdominal wall (it detects closure of the cardiac valves) or;
2. *The internal monitor.* An electrode is attached to the fetal scalp which will record the fetal ECG (electrocardiography). Internal monitoring cannot be used unless the cervix is dilated and the membranes are ruptured to allow replacement of the scalp electrode. The external monitor is without hazard but the internal monitor carries the risk of infection of scalp or amnion, perforation of uterus or placenta or fetal damage due to misapplication if fetal malpresentation was not recognized. However, internal monitor is recommended because it is more accurate and avoids the artifacts caused by fetal movement. The uterine contractions are recorded simultaneously with the FHR (Cardiotocography or CTG). Tocography may be external (tocometer is attached to abdominal wall) or internal by the use of an intrauterine catheter. The latter is more accurate because it measures the intrauterine pressure which accompanies the uterine contractions, while external tocography records the movement of the anterior abdominal wall occurring with uterine contractions.

Indications for Intrapartum Continuous Fetal Monitoring

The presence of a high risk factor as small-for-dates fetus, preterm labour, post-maturity, diabetes, hypertension, previous caesarean delivery, induction of labour with oxytocin, meconium stained amniotic fluid and abnormal FHR detected by fetoscope or Sonicaid. However, some centers apply continuous fetal monitoring for all patients in labour as 50% of cases of fetal asphyxia occur in absence of a high risk factor.

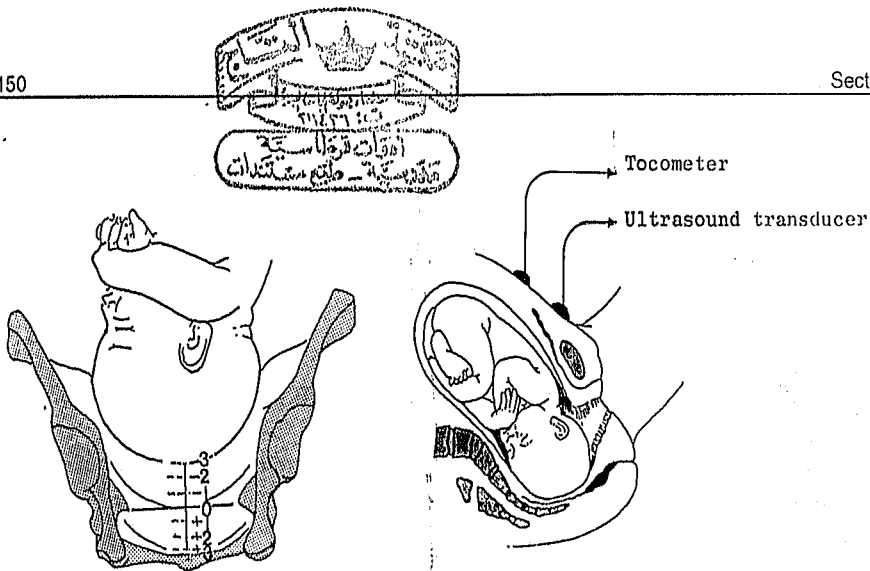


Fig. 69. Station of fetal head

Fig. 70. External monitoring of fetal heart rate and uterine contractions

Abnormal FHR Patterns

1. Tachycardia (more than 160 beats/min)

The causes are fetal distress, fetal anaemia, maternal anaemia, prematurity, maternal fever, chorioamnionitis, maternal or fetal hyperthyroidism, drugs as atropine and tocolytic agents. Sometimes paroxysmal attacks of tachycardia occur without a cause.

2. Bradycardia (less than 120 beats/min)

Fetal distress is present when FHR is less than 120/min. Bradycardia may also result from congenital fetal heart block and beta blockers given to the mother.

3. Absence of beat-to-beat variation

Normally, the fetal heart changes its rate by 5 beats or more every minute. This occurs in response to nervous and hormonal factors. If the rate of variation is less than 5 beats per minute, the trace appears flat. The commonest cause is hypoxia but can also occur during fetal sleep and with drugs which sedate the mother as Valium and barbiturates.

4. Early decelerations (Type I dips)

Due to compression of fetal head during uterine contraction leading to increased intracranial tension. This leads to vagal stimulation and slowing of the heart. Slowing starts with the onset of contraction (early onset), the lowest point corresponds with the peak of contraction and there is rapid recovery towards the base line at the end of contraction (reverse image to the contraction). It does not indicate fetal distress.

5. Late decelerations (Type II dips)

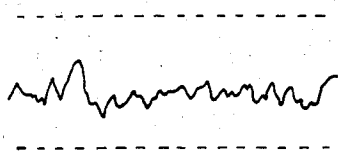
It indicates fetal hypoxia and distress due to reduced placental blood flow caused by uterine contraction. Deceleration begins at or near the peak of contraction (late onset) and recovery to base line begins when the contraction is completed.

6. Variable decelerations

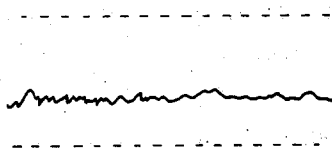
Due to intermittent compression of umbilical cord by fetal head. Slowing is variable in its degree, duration and relationship to uterine contractions, as it may occur early or late in relation to contraction. Fetal distress is present if this deceleration is severe (slowing below 70 per minute) and persistent (lasting over 30 seconds with each contraction for 30 minutes or more).

7. Prolonged decelerations

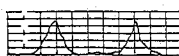
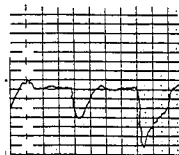
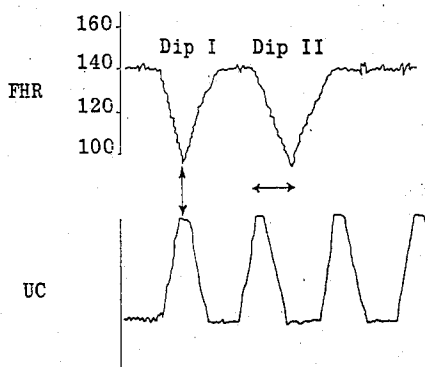
These are isolated decelerations which last longer than 60–90 seconds. Causes include tetanic uterine contractions, placental abruption, or umbilical cord prolapse (continuous not intermittent compression).



Normal trace of fetal heart rate



Absence of beat-to-beat variation



Variable deceleration

Fig. 71. Fetal heart tracing during labour. FHR = fetal heart rate. UC = uterine contractions

8. Sinusoidal pattern

It indicates fetal anaemia. Also caused by narcotics. If present and is unrelated to the administration of narcotics a fetal scalp blood is examined for pH and haematocrit to detect fetal anaemia. If values are abnormal, immediate delivery is carried out. It is characterized by a normal base line with regular sine-wave oscillations above and below the base line occurring 2 to 8 times per minute.

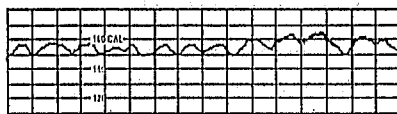


Fig. 71-2. Sinusoidal pattern.

Notes:

1. All the abnormal patterns mentioned except early deceleration can indicate fetal distress.
2. If abnormal FHR pattern develops, the patient is asked to lie on her left side, oxygen is given and oxytocin infusion is stopped. These measures may correct the abnormal pattern and prevent unnecessary intervention. If the abnormal pattern persists, fetal blood sampling for pH can be done to avoid unnecessary caesarean section.
3. In late pregnancy when the woman turns from supine position to her left side the cardiac output increases 20%, but only 10% when she lies on her right side.
4. Fetal distress is due to fetal hypoxia with or without acidosis.

IMPROVING DETECTION OF THE COMPROMISED FETUS

Electronic monitoring of the fetal heart is associated with high "false-positive" results and so many fetuses with abnormal fetal heart rate pattern will be normal at birth. This leads to increased rates of caesarean and instrumental vaginal delivery. To reduce the rate of high "false-positive" results, other methods are used to evaluate the fetal status when the FHR pattern is abnormal. These include fetal scalp and acoustic stimulation, fetal blood sampling, and fetal pulse oximetry.

A. Fetal Scalp and Acoustic Stimulation

Stimulation of the fetal scalp is done digitally at the time of vaginal examination or with an Allis clamp. When accelerations occur in the FHR, fetal acidosis is excluded. It is also excluded when accelerations follow fibroacoustic stimulation.

B. Fetal Blood Sampling

Fetal hypoxia may result in fetal acidosis. Blood (0.2 ml) is collected by making a small cut in the fetal scalp using the amnioscope. If the pH is 7.20 or less, it indicates acidosis and the fetus should be delivered immediately. If the pH is 7.25 or greater (normal 7.25–7.35) labour can be allowed to continue. Intermediate values require repeated sampling. The new scalp electrode can record the fetal pH continuously.

C. Fetal Pulse Oximetry

The sensors are placed on a flat surface such as the fetal cheek to measure the arterial oxygen saturation.

OBSERVATION FOR THE PRESENCE OF MECONIUM IN AMNIOTIC FLUID

The passage of meconium in cephalic presentation indicates fetal distress. Hypoxia leads to increased intestinal movements and relaxation of anal sphincter. Internal fetal monitoring is indicated in this situation because in some cases meconium is present without fetal distress. When meconium is associated with abnormal FHR pattern, labour is terminated immediately or fetal blood sampling for pH is done to avoid unnecessary caesarean section.

Note: Meconium consists of desquamated epithelial cells from the fetal gastrointestinal tract, mucus, secretions from the gastrointestinal tract (secretions from stomach, pancreas, intestine, and bile), fetal epidermal cells and lanugo hair that have been swallowed with amniotic fluid. Meconium is semisolid and brownish-green in colour due to bile pigments.

HIGH-RISK PREGNANCY

It is a pregnancy in which there is an increased risk to the mother, fetus or neonate. At least 20% of all pregnancies are high risk. The high risk situation includes:

I. Factors in the History

- Maternal age below 20 and above 35 years.
- Grand multipara.
- Drug addiction, alcoholism or heavy smoking.
- Bad obstetric history: History of recurrent abortions, preterm labour, stillbirth and neonatal death.
- History of large (4 kg or more) or malformed baby.
- Exposure to teratogenic agents (drugs, Rubella, etc.)
- Erythroblastosis fetalis.
- History of trauma or disease affecting the bony pelvis.
- Presence of scar in the uterus.
- Anomalies of the genital tract as patulous cervix and bicornuate uterus, fibroids and ovarian cyst.

II. Maternal Diseases

Pre-eclampsia and eclampsia, diabetes, chronic hypertension, pulmonary, cardiac, renal, gastrointestinal, liver and endocrine diseases. Moderate and severe anaemia. Malignancy and blood diseases.

III. Feto-Maternal Factors

Multiple pregnancy, preterm labour, premature rupture of membranes, intrauterine growth restriction, postterm pregnancy, malpresentations, polyhydramnios, oligohydramnios, antepartum haemorrhage and cephalopelvic disproportion.

These cases are examined more frequently during the antenatal period and preferably in a specialized high risk clinic and delivery should be in hospital under strict supervision.

CONTROL OF PAIN DURING LABOUR

ANATOMY OF PAIN

Pain in the first stage of labour is due to uterine contractions and dilatation of cervix. Pain impulses pass from the body of uterus along sympathetic nerve fibres to enter spinal segments T11, T12 and L1. Pain from cervix passes along parasympathetic nerve fibres which accompany the 2nd, 3rd and 4th sacral nerves. In the first stage the intrauterine pressure is about 10 mmHg between contractions and 50 mmHg during contractions. Patient feels pain when the pressure exceeds 25 mmHg.

Pain in the second stage is due to uterine contractions and dilatation of the vagina and stretching of the vulva and perineum by the presenting part. Pain impulses are carried by pudendal nerve and to a small extent by ilioinguinal nerve, genitofemoral nerve and posterior cutaneous nerve of the thigh. Pudendal nerve is formed from S2, S3 and S4.

I. ANALGESICS

1. Narcotics

- A) *Pethidine*: A synthetic drug. It is a sedative, analgesic and antispasmodic. The best analgesic drug. Dose is 50–100 mg intramuscularly. Action lasts two hours so contraindicated if delivery is expected to occur within two hours because it depresses the fetal respiratory center. The antidote is naloxone (Narcan) which is injected into the umbilical vein, IM or subcutaneously (10 micrograms/kg). Pethidine sometimes has an emetic effect.
- B) *Morphine*: Dose is 10–15 mg IM. Action lasts 4 hours. It depresses the fetal respiratory center. It is not used during labour unless the fetus is dead.

2. Tranquilizers

- A) *Sparine* (promazine hydrochloride): It is a tranquilizer and antiemetic. It potentiates the action of pethidine and other analgesic drugs. It does not depress the fetal respiratory center. Dose is 50 mg IM.
- B) *Valium* (diazepam): It can be used in the first stage of labour. Dose 5–10 mg IM every 4 hours. It may cause fetal hypotonia and hypothermia. Not recommended in the preterm baby because of the risk of hyperbilirubinaemia and kernicterus.

3. Inhalation Analgesia

- A) *Nitrous oxide and oxygen*: A special machine (Entonox) delivers a mixture of nitrous oxide 50% and oxygen 50% to produce analgesia. The patient inhales the mixture once she feels that a contraction is coming on. It is relatively safe for the mother and fetus. However, inhalation analgesia should not be used for more than 3 hours otherwise we get depression of fetal respiratory center.
- B) *Trilene and air* using the small "Cyprane apparatus". Trilene is a volatile liquid which is no more used because of toxicity.

II. ANAESTHETICS

1. General Anaesthesia

Nitrous oxide and oxygen is a safe anaesthetic (60% nitrous oxide and 40% oxygen). As it produces little muscular relaxation it is not suitable for intrauterine manipulations as internal podalic version and reduction of inverted uterus. In these cases it is combined with halothane (0.5%).

2. Spinal Anaesthesia

It is difficult to perform in pregnancy due to enlargement of the abdomen and may cause spinal shock. The anaesthetic drug is injected into the subarachnoid space.

3. Epidural Anaesthesia

- A) *Lumbar route*: The anaesthetic drug (lignocaine 1% or Marcaine 0.5%) is injected into the epidural space between 3 and 4 lumbar vertebrae.
- B) *Caudal route*: The anaesthetic drug is injected through the sacral hiatus into the epidural space.

Epidural anaesthesia is the most effective method of pain relief in labour and does not affect the fetus. However, the incidence of forceps delivery is increased as it abolishes the perineal reflex and hence the desire to bear down resulting in prolonged second stage of labour. It also leads to atony and relaxation of the pelvic floor and may lead to failure of internal rotation of the head.

4. Paracervical Block

The drug (lignocaine 1%) is injected into the paracervical tissues on both sides of the cervix by inserting a needle through the lateral vaginal fornix on each side 2 cm lateral to the cervix and 1–2 cm deep to block the paracervical plexus of nerves. Inject 5 ml on each side. The effect lasts 1–2 hours and the injection can be repeated. Used when the cervix is dilated more than 4 cm. Transplacental transfer of the drug may cause fetal bradycardia or

even fetal death so the method is rarely used. Bradycardia results from vasoconstriction of uterine arteries.

5. Pudendal Nerve Block

Ten ml of 1% lignocaine solution are injected just below the ischial spine on both sides using the vaginal route. It can be used instead of general anaesthesia for mid forceps delivery as in case of congestive heart failure and for low forceps delivery and vacuum extraction. Failure rate is common (10–15%).

6. Perineal Infiltration

It is used for episiotomy and repair of a perineal tear. The perineum is infiltrated along the line of incision using 10 ml of 1% lignocaine solution.

III. ANALGESIA WITHOUT THE USE OF DRUGS

1. *Antenatal instruction classes and exercises.* The patient is told about the stages of labour in simple terms. She should visit the labour ward and if possible meet the staff who will look after her there. Respiratory and muscle relaxation exercises spare oxygen for the contracting uterus thus reducing uterine ischaemia responsible for pain.
2. *Acupuncture:* It may relieve pain in some women.
3. *Underwater labour.* Tried in certain centers. The woman uses the swimming pool during the first stage of labour. It is believed that immersion in water reduces sensory stimulation and catecholamines secretion and so pain is relieved.

SUMMARY

1. First stage of labour. Pethidine, sparine or a combination can be given and repeated if necessary.
2. Second stage. Inhalation of nitrous oxide and oxygen.
3. Local infiltration anaesthesia for episiotomy and repair of a perineal tear.
4. Nitrous oxide and oxygen for obstetric operations.
5. Lumbar epidural anaesthesia. It relieves pain throughout labour and also for obstetric operations.

UTERINE STIMULANTS (ECBOLICS – OXYTOCICS)

I. POSTERIOR PITUITARY EXTRACT

Pituitrin is the posterior pituitary extract; it has two fractions; oxytocin which is mainly uterine stimulator and vasopressin which is mainly vasopressor and antidiuretic. Pitocin is the pure natural oxytocin while Syntocinon is a synthetic oxytocin.

Action

Pitocin or Syntocinon only affects the pregnant uterus. It increases the frequency, strength and duration of uterine contractions. Action lasts about half an hour.

Indications

1. Management of certain types of abortion as inevitable, incomplete and missed abortion to help the uterus to expel its contents.
2. Postabortive bleeding.
3. During evacuation of hydatidiform mole to avoid perforation of uterus and excessive bleeding.
4. Induction of labour.
5. Oxytocin stress test to assess fetal well-being.
6. Uterine inertia during labour.
7. Abruptio placentae (accidental haemorrhage).
8. In the management of third stage of labour.
9. Postpartum haemorrhage.
10. During caesarean section. It is injected intravenously or directly into the uterine wall after extraction of the fetus to hasten separation of the placenta and reduce bleeding.
11. Breast engorgement: oxytocin nasal drops or spray or buccal tablet at time of breast feeding to increase milk flow.

Contraindications

1. Obstructed labour, otherwise rupture of uterus will occur.
2. Malpresentation as transverse or oblique lie.
3. Scar in the uterus (relative contraindication).
4. The grand multipara (delivered 5 times or more, relative contraindication).
5. Fetal distress.
6. Placental insufficiency.

Complications

Uterine hyperstimulation, rupture of uterus, cervical lacerations, contraction ring, secondary uterine inertia with subsequent postpartum haemorrhage, fetal asphyxia, amniotic fluid embolism, coronary spasm and shock if pituitrin is used. Water intoxication if a large dose is given for a prolonged period because of the antidiuretic effect. Maternal hypotension occurs with rapid IV injection. Neonatal hyperbilirubinaemia and jaundice. Oxytocin competes with bilirubin for binding with albumin.

Methods of Administration

1. *Orally*: A tablet is placed between the gum and cheek and left to dissolve slowly in 30 minutes (Sandopart). It can be repeated every 1/2 hour.
2. *Intramuscularly*, but this is avoided before delivery of the fetus.
3. *Intravenously* by the drip method and this is the best.
4. *Intrauterine* during caesarean section.
5. *Nasal* spray or drops.

II. ERGOT ALKALOIDS

Ergot is a plant containing 4 alkaloids, the most important is ergometrine (ergonovine).

Action

It causes a prolonged uterine spasm. The action lasts about 4 hours. It should never be used before delivery of the fetus as it may lead to tonic contraction of uterus causing fetal asphyxia and rupture of uterus.

Indications

1. Management of certain types of abortion as inevitable and incomplete abortion.
2. Postabortive bleeding.
3. In the management of third stage of labour.
4. Postpartum haemorrhage.
5. During caesarean section.
6. Subinvolution of uterus.

Contraindications

1) Before delivery of the fetus; (2) hypertension due to any cause as it raises the blood pressure; (3) heart disease.

Methods of Administration

1. *Orally* in the form of tablets or drops. The dose is 1 mg and action starts after 7 minutes.
2. *Intramuscularly*, 0.5 mg and action starts after 3.5 minutes.
3. *Intravenously*, 0.25 mg and action starts in less than one minute (45 seconds).

III. PROSTAGLANDINS

These are unsaturated fatty acids found in many tissues and tissue fluids. There are 9 naturally occurring groups of prostaglandins which have been labelled A-I and in each group there are 3 main compounds depending on whether the molecule contains 1, 2 or 3 double bonds. Some of the compounds have isomers in which case the naturally occurring compound is given the suffix α . Depending on which prostaglandin is used and on which

system, these compounds can be muscle relaxants or stimulants, vasodilators or vasoconstrictors. Prostaglandins E_2 , E_1 analogue and $F_{2\alpha}$ are strong oxytocics. The drugs can be given orally, intramuscularly, intravenously, intramyometrial, intra-amniotic, extra-amniotic or as vaginal tablets or gel. Misoprostol which is prostaglandin E_1 analogue can be given orally, vaginally, and rectally. These agents do not cause hypertension, and so can be used in hypertensive women.

Action in Obstetrics

1. Ripening of the cervix (to make it short, soft, and partially dilated). Prostaglandins are the best agents for induction of labour if the cervix is unripe.
2. Stimulation or initiation of uterine contractions at any stage of pregnancy.

Indications

1. Induction of abortion.
2. Induction of labour.
3. Treatment of atonic postpartum haemorrhage.

Complications

Nausea, vomiting, diarrhoea, hyperpyrexia, bronchospasm, bradycardia, uterine hyperstimulation, cervical lacerations, and rupture of uterus.

Advantages of Prostaglandins Over Oxytocin Infusion for Labour Induction

1. Prostaglandins are used when the cervix is unripe, while oxytocin infusion is not used in this situation.
2. Reduced incidence of neonatal hyperbilirubinaemia and jaundice which may occur with oxytocin infusion.
3. Maternal water intoxication may occur with oxytocin infusion when a large dose is given for a long period.

Notes:

- *Intra-amniotic*: Solution is injected into the amniotic sac through the abdominal wall.
- *Extra-amniotic*: With the membranes intact a catheter is passed along the cervical canal into the uterus to inject the solution. These methods are used to induce abortion or labour when the fetus is dead or malformed.
- *Intramyometrial*: Needle passed through the abdominal wall to treat atonic PPH.
- Prostaglandins are generally contraindicated in women with asthma, pulmonary hypertension, ischaemic heart disease, and epilepsy.

TOCOLYTIC DRUGS

Drugs which inhibit uterine contractions to prevent preterm labour include beta-sympathomimetic drugs, prostaglandin-inhibiting agents, magnesium sulphate, calcium channel blockers, and nitroglycerin. Tocolytics are not given after 34 weeks of pregnancy as the incidence of respiratory distress syndrome will be low (2%–7%).

1. BETA-MIMETIC (SYMPATHOMIMETIC) DRUGS

Cause relaxation of the smooth muscle fibres by stimulating the beta-adrenergic receptors present on the cell membrane. These drugs can inhibit premature uterine contractions and are effective if the membranes are intact and the cervix is less than 3 cm dilated.

Complications

a) Maternal

General: Headache, nausea, vomiting, flushing, sweating, anxiety, and tremors.

Cardiac: Tachycardia, arrhythmias, palpitations, myocardial ischaemia and chest pain, heart failure, systolic hypertension and diastolic hypotension (due to relaxation of smooth muscle fibres in the wall of blood vessels), and pulmonary oedema.

Metabolic: Hyperglycaemia, hyperinsulinaemia (the drugs stimulate the pancreas to secrete both glucagon and insulin), acidosis, hypokalaemia, hypocalcaemia, antidiuretic effect, and increased thyroid activity.

Others: Paralytic ileus, skin rash, and pruritus.

b) Fetal

Tachycardia, arrhythmias, loss of beat-to-beat variation, myocardial ischaemia and heart failure, hypotension, hyperglycaemia while the fetus is in utero and hypoglycaemia in the newborn, hypokalaemia, hypocalcaemia, paralytic ileus, and hyperbilirubinaemia.

Pulmonary Oedema: It occurs in about 5% of cases. The drugs have antidiuretic effect leading to retention of sodium with increased plasma volume. Predisposing factors include multiple pregnancy, heart disease, pre-eclampsia, corticosteroids, and intravenous saline solution.

Contraindications

(1) Heart disease; (2) hypertension; (3) hyperthyroidism; (4) antepartum haemorrhage (dilatation of uterine arteries can increase the bleeding); (5) uncontrolled diabetes.

Administration

Ritodrine (Yutopar) 50 mg in 500 ml 5% Dextrose solution. Start by 50 micrograms/minute and increase by 50 micrograms every 10 minutes until uterine contractions cease. The infusion should be continued for at least 12 hours after cessation of contractions. The treatment is then maintained by giving oral tablets; one tablet (10 mg) every 6 or 8 hours (after meal to reduce absorption and side effects). The dose is increased or decreased according to response and side effects. When giving the infusion, the maternal pulse and blood pressure and fetal heart rate should be recorded before increasing the dose. Infusion is stopped if maternal pulse >120 bpm, systolic blood pressure >180 mmHg, diastolic pressure <40 mmHg, fetal heart rate >200 bpm, or persistent chest pain.

2. PROSTAGLANDIN INHIBITORS

The most commonly used drug is indomethacin (Indocid). It is given orally but usually rectally to avoid gastric irritation. The oral dose is 25 mg every 6 hours or a suppository 100 mg is inserted twice daily. It is given for 3 or 4 days but not more to avoid fetal complications, but can be repeated after 5 days. Fetal complications include: (a) premature closure of the ductus arteriosus leading to pulmonary hypertension, heart failure and even death, (b) necrotizing enterocolitis, and (c) oligohydramnios due to reduced fetal renal blood flow, and increased absorption of amniotic fluid by the bronchial mucosa as the drug increases fetal respiratory movements, and it has antidiuretic effect. The drug is contraindicated after 34 (or 32) weeks of pregnancy as the risk of closure of the ductus arteriosus (5-10%) increases after 34 (or 32) weeks. Prostaglandin E2 and prostacyclin are responsible for keeping the ductus arteriosus patent.

3. MAGNESIUM SULPHATE

It is less effective than beta-mimetic drugs, and is used when these drugs are contraindicated or cause toxicity. It is given as enteric coated magnesium gluconate tablets or by intravenous infusion in the same way as mentioned in eclampsia. Complications include cardiac and respiratory depression and even arrest, hypotension, paralytic ileus, and pulmonary oedema. The antidote is calcium which is given intravenously as 10 ml of a 10% calcium gluconate solution (1 gm). The only absolute contraindication is myasthenia gravis. Magnesium sulphate inhibits uterine activity by displacing intracellular calcium. To maintain tocolytic effect, magnesium gluconate tablets are given as 1 g every 2-4 hours.

4. CALCIUM CHANNEL BLOCKERS

Calcium antagonists can inhibit premature uterine contractions with few side effects.

Nifedipine is given as 10 mg orally or sublingually and repeated every 20 minutes up to 4 doses or until uterine contractions subside. The maintenance therapy is 10-20 mg

given orally or sublingually every 6 hours. It can cause marked hypotension. The drugs act by preventing the passage of calcium ion into the cells.

5. NITROGLYCERIN

Administered in the form of abdominal patches. Usually one statim (10 mg) and repeated in one hour if uterine contractions continue. It releases 10 mg in 24 hours.

ABNORMAL LABOUR

MALPRESENTATIONS

OCCIPITO-POSTERIOR POSITION (O.P.)

Definition

It is a vertex presentation in which the fetal back is directed posteriorly. The occiput is felt towards the sacro-iliac joint.

Incidence

It occurs in about 10% of vertex presentations. Right occipito-posterior is commoner than left occipito-posterior (4:1) because:

1. The right oblique diameter of the pelvis is slightly longer than the left due to the more frequent use of the right leg.
2. The left oblique diameter is reduced by the presence of the pelvic colon.
3. Dextrorotation of uterus. This favours O.P. if the back is on the right side.

Aetiology

1. Special types of pelvis; as android or anthropoid pelvis. This is the commonest cause.
2. Maternal lumbar kyphosis, the convexity of the back of the fetus fits into the concavity of the lumbar kyphosis.
3. Anterior insertion of the placenta; the concavity of the front of the fetus fits into the convexity of the placenta, also the fetus usually faces the placenta.
4. Other causes of malpresentation in general as multiparity with lax abdominal and uterine wall, placenta praevia, multiple pregnancy and polyhydramnios.
5. Idiopathic: In about 20% of cases no cause is found.

Diagnosis

I. During Pregnancy

A) Abdominal Examination

1. Inspection

- a) The abdomen is flat below the umbilicus (due to absence of the contour of the fetal back).
- b) A transverse groove may be seen below the umbilicus (corresponds to the groove of the neck).

c) Fetal movements may be seen on both sides of the middle line.

2. *Palpation*

a) Fundal level: It corresponds to the period of amenorrhoea.

b) Fundal grip: The breech is felt occupying the fundus.

c) Umbilical grip: The back is felt with difficulty in the flank away from the midline. The limbs are felt on both sides of the midline. In direct occipito-posterior the back is not felt at all. The subumbilical groove may be felt.

d) First pelvic grip: The head is usually not engaged. It feels small because the hand grasps the bitemporal diameter (8 cm) which is smaller than the biparietal diameter (9.5 cm). The head tends to recede away from the examining fingers.

e) Second pelvic grip: The head is deflexed in most of the cases.

3. *Auscultation*

The fetal heart sounds are heard away from the mid-line near the anterior superior iliac spine. In direct occipito-posterior the FHS may be heard in the middle line between the umbilicus and symphysis pubis.

B) Sonography

This confirms the diagnosis when it is clinically difficult as in case of obesity.

II. *During Labour*

A) Abdominal examination; as before.

B) Vaginal examination; this will reveal

1. Slow dilatation of the cervix, a voluminous bag of forewaters, liability to premature rupture of membranes and prolapse of cord as the head is not fitting well against the lower uterine segment.
2. When the cervix is sufficiently dilated especially after rupture of the membranes, the anterior fontanelle is felt anteriorly towards the obturator foramen, while the posterior fontanelle is felt posteriorly towards the opposite sacro-iliac joint. When the fontanelles are masked by a caput succedaneum palpation of the ear helps diagnosis. The helix is towards the occiput, the tragus is towards the face, or we palpate the cervical spine.

Deflexion of the Head

In most cases of occipito-posterior the head is deflexed because:

1. The convexity of the maternal lumbar spine is against the convexity of the fetal back. This causes extension of the back of the fetus and deflexion of its head.

2. The biparietal diameter of the head descends in the narrow sacro-cotyloid diameter of the pelvis (9.5 cm), while the smaller bitemporal diameter descends into the wide oblique diameter. This leads to deflexion of the head.
3. As with any malpresentation there is liability to premature rupture of the membranes and drainage of amniotic fluid, so the uterus acts directly on the fetus squeezing it, causing straightening of the spine and deflexion of the head.

As a result of deflexion, the head descends by a long diameter, the occipito-frontal (11.5 cm) and so the head is delayed during its descent. Also the deflexed head will not fit well against the lower uterine segment and this predisposes to premature rupture of membranes, and prolapse of cord. Also the uterine contractions tend to be weak, infrequent and irregular due to interference with reflex polarity as the head is not fitting well against the lower uterine segment (these effects can happen with any malpresentation).

Note: Reflex polarity means pressure by the presenting part on the lower uterine segment and cervix reflexly stimulates the uterus to contract.

Mechanism of Labour

1. Normal Mechanism

This occurs in 90% of cases. As the head descends it becomes completely flexed so the occiput meets the pelvic floor first; therefore, it rotates forwards $\frac{3}{8}$ of a circle (long anterior rotation). This is accompanied by internal rotation of the anterior shoulder $\frac{2}{8}$ of a circle behind the symphysis pubis so that the biacromial diameter occupies the opposite oblique diameter. The head is then delivered by extension as in occipito-anterior position.

2. Abnormal Mechanism (10%)

- a) When deflexion of the head is slight the occiput rotates forwards $\frac{1}{8}$ of a circle (short anterior rotation) and the head becomes arrested in the transverse diameter of the pelvis. This is called deep transverse arrest (1%).
- b) When deflexion is moderate the occiput and sinciput meet the pelvic floor at the same time and so no rotation occurs and the head remains in the oblique diameter of the pelvis, this is called persistent occipito-posterior (3%).
- c) With severe degree of deflexion the sinciput meets the pelvic floor first so the sinciput rotates forwards $\frac{1}{8}$ of a circle and the occiput rotates backwards leading to direct occipito-posterior or face to pubis (6%). In 50% of these cases delivery occurs spontaneously. The head descends until the root of the nose becomes fixed below the symphysis pubis, the occiput is delivered by flexion and then the face by extension. In 50% of cases the head is arrested and forceps is needed. Episiotomy must be done in

these cases because there is liability to perineal lacerations due to: (1) the vulva is distended by a long diameter; the occipito-frontal (11.5 cm); (2) the bulky occipital region of the head is posterior, and so it markedly stretches the perineum.

In deep transverse arrest and persistent occipito-posterior labour become obstructed.

Causes of Failure of Internal Rotation of the Head (Abnormal Mechanism)

1. Deflexion of the head, which is the commonest cause.
2. Weak uterine contractions.
3. Weak or rigid pelvic floor.
4. Contracted pelvis which mechanically interferes with rotation.
5. Premature rupture of the membranes and drainage of the liquor amnii. This interferes with rotation of the head and shoulders.
6. Less important factors include full bladder or rectum, placenta praevia, pelvic tumour and large head.

Management

I. During Pregnancy

The mother is encouraged to lie on her side opposite to that where the fetal back is present so that by gravity the back and occiput may rotate forwards.

II. During Labour

A) First Stage

The same management as in normal labour. However, the patient is kept in bed. No walking is allowed to avoid premature rupture of membranes and prolapse of cord as the deflexed head is not fitting well against the lower uterine segment.

B) Second Stage

1. When the membranes rupture vaginal examination is performed to estimate the degree of cervical dilatation, to exclude prolapse of cord and to confirm the diagnosis.
2. The head is given a chance to rotate forwards. Oxytocin drip is given to help rotation if the uterine contractions are weak, which is frequent in these cases. However, if the second stage is prolonged (more than 1 hour in the primigravida and 1/2 hour in the multigravida) or there is fetal or maternal distress, interference is indicated.

Management of Persistent Occipito-Posterior

- a) If the fetal head is not engaged, caesarean section is done.
- b) If the head is engaged the treatment will be one of the following lines:

1. Manual rotation and delivery by forceps as occipito-anterior: Under general anaesthesia the head is grasped by the hand and is rotated by four movements: disimpaction, increased flexion, anterior rotation of the occiput and rotation of the anterior shoulder towards the midline by the abdominal hand. After rotating the head $3/8$ of a circle the forceps is applied as usual. This method is not used except in few centers.
2. Rotation and extraction by Kielland forceps. Not used except in few centers.
3. The vacuum extractor may be used for delivery. The head rotates during extraction.
4. Caesarean section: the indications are:
 - Failure of the above measures.
 - Contracted pelvis.
 - Elderly primigravida.
 - Caesarean section is done in every case, as it is safer for the mother and fetus than manual rotation and delivery by forceps, or rotation and extraction by Kielland forceps.
5. Craniotomy: May be done when the fetus is dead and labour is obstructed. However, caesarean section can be done as it is safer for the mother.

Management of Deep Transverse Arrest

The same as persistent occipito-posterior. In this case the head is engaged.

Management of Direct Occipito-Posterior

In about 50% of cases the fetus is born spontaneously as face to pubis. However, if labour is prolonged or if there is fetal or maternal distress the forceps is applied. Episiotomy is needed in these cases because there is liability to perineal lacerations.

Postural Technique in Occipito-Posterior

During labour the patient is encouraged to lie on her side opposite to that where the fetal back is present. This helps forward rotation of the back and occiput and makes uterine contractions stronger and less frequent.

Complications of Occipito-Posterior

A) Maternal Complications

(1) Premature rupture of membranes and prolapse of cord; (2) prolonged labour; (3) obstructed labour as in case of deep transverse arrest; (4) uterine inertia due to absence of reflex polarity; (5) postpartum haemorrhage due to inertia; (6) liability to puerperal sepsis; due to prolonged labour; premature rupture of membranes and increased incidence of interference; (7) perineal lacerations when the fetus is born as face to pubis.

B) Fetal Complications

1. Asphyxia due to prolonged labour or compression of prolapsed cord.
2. Operative birth injuries.

FACE PRESENTATION

Definition

It is a cephalic presentation in which the head is completely extended, so the fetus presents by the face.

Incidence

About one in 500 labours.

Positions

The denominator is the chin (mentum). There are 8 positions; right & left mento-anterior, right & left mento-posterior, right & left mento-transverse, direct mento-anterior and direct mento-posterior. Mento-anterior are more common than mento-posterior positions because most cases of face presentation result from extension of the head in occipito-posterior.

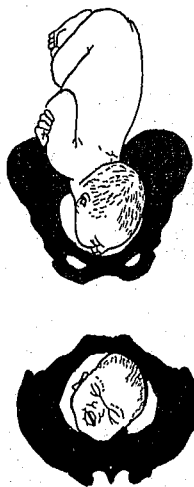


Fig.72. Right mento-posterior

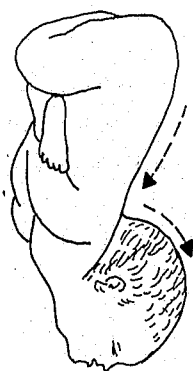


Fig. 73. Deep groove between head and back

Aetiology

A) Maternal Causes

1) Pelvic contraction; (2) pelvic tumour; (3) placenta praevia; (4) polyhydramnios; (5) multiparity with lax abdominal and uterine wall; (6) marked lateral obliquity of the uterus.

B) Fetal Causes

1. Anencephaly (absence of the vault of the skull and cerebral hemispheres).
2. Dolichocephalus (long antero-posterior diameter of the head).
3. Tumours of the neck, as congenital goitre or cystic hygroma.
5. Loops of the umbilical cord around the neck (nuchal cord).
4. Spasm of the extensor muscles of the neck. This may be a result and not a cause of face presentation.
6. Intrauterine fetal death due to lack of tone of flexor muscles.

Types

1. Primary face presentation. It is face presentation detected during pregnancy. Very rare, mostly due to fetal causes.
2. Secondary face presentation. This means that the head becomes extended during labour, mostly due to maternal causes.

Diagnosis

I. During Pregnancy

A) Abdominal Examination

1. Inspection

- a) In mento-anterior the fetal movements may be seen on both sides of the midline.
- b) In mento-posterior positions a transverse groove may be seen below the umbilicus which corresponds to the groove between the fetal head and back.

2. Palpation

- a) Fundal level: It corresponds to the period of amenorrhoea.
- b) Fundal grip: The breech occupies the fundus.
- c) Umbilical grip: In mento-anterior position, it is difficult to feel the back, but the limbs are easily felt on both sides of the midline. In mento-posterior position, the sub-umbilical groove is felt.

- d) First pelvic grip: The head is not engaged.
- e) Second pelvic grip: The occiput is felt at a higher level than the sinciput.

3. Auscultation

In mento-posterior position, the fetal heart sounds are difficult to hear, while in mento-anterior they are easily heard below the umbilicus because the chest is thrown forwards against the abdominal wall.

B) Sonography

This confirms the diagnosis and excludes fetal malformation.

II. During Labour

A) Abdominal examination; as before.

B) Vaginal examination; this will reveal:

1. Slow dilatation of the cervix, a voluminous bag of forewaters, liability to premature rupture of membranes and prolapse of cord as the head is not fitting well against the lower uterine segment.
2. When the cervix is sufficiently dilated especially after rupture of the membranes, the bony landmarks of the face are felt: the orbital margins, the nose, the alveolar margins and the chin. Later in labour, there is oedema or tumefaction of the face. The face may be mistaken for frank breech.

Mechanism of Labour

I. Mento-anterior

The head descends with the submento-bregmatic diameter (9.5 cm). There is descent, engagement, increased extension of the head so the chin meets the pelvic floor first and rotates forwards $1/8$ of a circle. With further descent the submental-region appears below the symphysis pubis and the head is delivered by flexion. This is followed by restitution and external rotation of the chin as in vertex presentation.

II. Mento-posterior

- a) In two-thirds of cases the chin rotates forwards $3/8$ of a circle and is delivered as mento-anterior.
- b) In $1/3$ of cases the chin rotates forwards $1/8$ of a circle and this is known as deep transverse arrest of the face, or it does not rotate at all and remains as persistent mento-

posterior, or the chin rotates backwards $1/8$ of a circle towards the concavity of the sacrum, this is direct mento-posterior. In all these cases labour becomes obstructed.

N.B.: In direct mento-posterior labour is obstructed because delivery needs extension of the head which is already fully extended.

Management

A) During the First Stage of Labour

The same management as in normal labour. However, the patient is kept in bed. No walking is allowed to avoid premature rupture of membranes and prolapse of cord.

B) During the Second Stage

1. When the membranes rupture, vaginal examination is performed to estimate the degree of cervical dilatation, to exclude prolapse of cord and to confirm the diagnosis.
2. In mento-anterior position, spontaneous delivery is allowed. If there is fetal or maternal distress or the second stage is prolonged, the forceps is applied if the head is engaged and caesarean section if not engaged.
3. In mento-posterior position we give a chance for the chin to rotate forwards. Oxytocin drip is given to help rotation if uterine contractions are weak which is frequent in these cases. However, if the second stage is prolonged or there is fetal or maternal distress, caesarean section is performed. When the fetus is dead, craniotomy may be done, however, caesarean section is safer for the mother.

Complications

Maternal and fetal complications as in occipito-posterior.

Fetal Mortality

About 9% due to fetal malformations incompatible with life as anencephaly, operative interference and fetal asphyxia due to prolonged labour, compression of prolapsed cord and oedema of the glottis.

N.B.: After delivery the parents are informed that congestion and oedema of the face of the baby will disappear spontaneously within a few days.

BROW PRESENTATION

Definition

It is a cephalic presentation with the head midway between flexion and extension.

Incidence

About 1 in 2000 labours.

Positions

The denominator is the frontal bone. There are 4 main positions; left fronto-anterior (LFA), RFA, RFP and LFP.

Aetiology

The same as in face presentation.

Diagnosis

I. During Pregnancy

A) Abdominal examination

It is difficult to diagnose brow presentation by abdominal examination. The condition is suspected when the occiput and sinciput are felt at the same level by the second pelvic grip, and when the fetal back is straight rather than curved.

B) Sonography:

This confirms the diagnosis and excludes fetal malformation.

II. During Labour

A) Abdominal examination; as before.

B) Vaginal examination; this will reveal

1. Slow dilatation of the cervix, a voluminous bag of forewaters, liability to premature rupture of membranes and prolapse of cord.
2. When the cervix is sufficiently dilated especially after rupture of the membranes the presenting part is felt, that is the two frontal bones with the frontal suture in between, the orbital margins, the anterior fontanelle on one side and the nose on the other side. The chin cannot be felt, if it is palpated, the presentation is face.

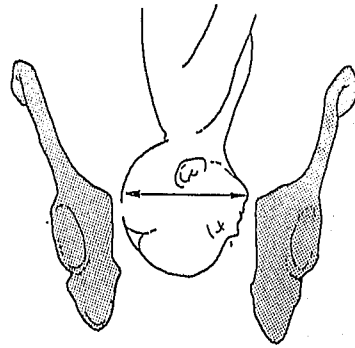


Fig. 74. Brow presentation with mento-vertical diameter presenting

Mechanism of Labour

There is no mechanism for delivery because the head descends by the mento-vertical diameter (13.75 cm) which is longer than any of the diameters of the pelvic inlet, so the head becomes arrested at the pelvic inlet and labour is obstructed. Sometimes a brow may be converted spontaneously into face (by extension) or vertex (by flexion) and this is followed by spontaneous delivery.

Management

If brow is detected in first stage of labour, it is considered transient brow and we wait for spontaneous conversion into face or vertex, keeping the mother and fetus under observation. Brow presentation detected late in the first stage or in the second stage is persistent brow and is treated by caesarean section. If the fetus is dead, craniotomy may be done, however, caesarean section is safer for the mother.

Note: The incidence of persistent brow is 10–20% of all cases of brow presentation.

BREECH PRESENTATION

Incidence

About 3.5% of all deliveries. The incidence is higher in preterm labours. Most cases of breech presentation undergo spontaneous cephalic version by the thirty-second week, and more cases by the thirty-six week.

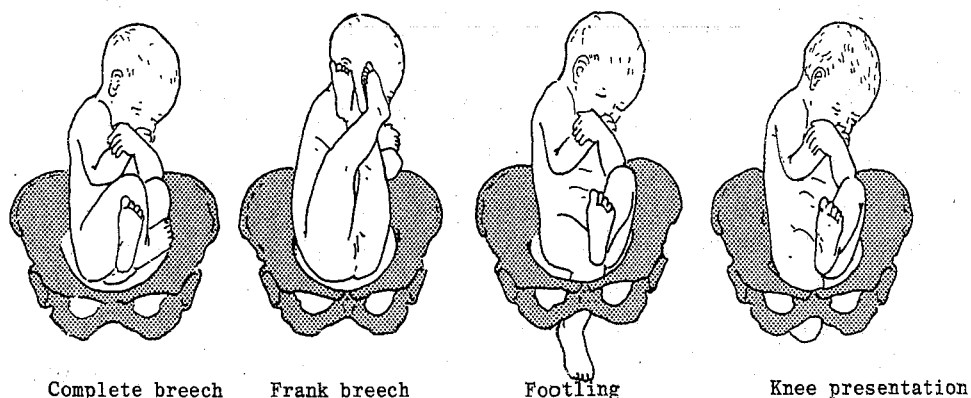


Fig. 75. Breech presentation

Types

1. **Complete breech:** One or both feet are present beside the buttocks. All the joints are flexed. It is more common in multipara.

2. *Incomplete breech*: It includes the following:

- a) *Breech with extended legs* (frank breech). The hip joints are flexed, the knee joints are extended with both feet in front of the head. It is more common in primigravidae.
- b) *Knee presentation*: One or both knees form the presenting part, due to extension of hip joints and flexion of the knee joints.
- c) *Footling presentation*: One or both feet form the presenting part, due to extension of the hip and knee joints.

Positions

The denominator is the sacrum. There are 8 positions; right & left sacro-anterior, right & left sacro-posterior, right & left sacro-transverse, direct sacro-anterior and direct sacro-posterior.

Anterior positions are commoner because the concavity of the front of the fetus fits into the convexity of the maternal lumbar spine.

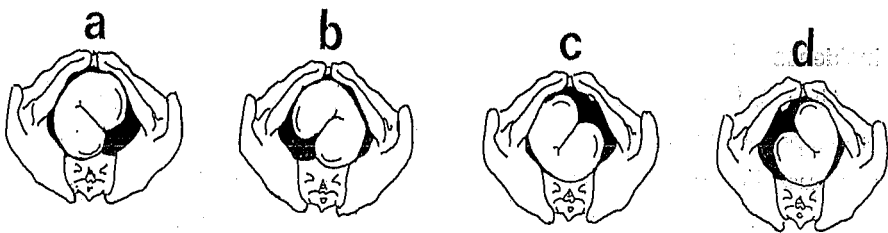


Fig. 76. (a) Right sacro-anterior, (b) left sacro-anterior, (c) right sacro-posterior, (d) left sacro-posterior

Aetiology

A) Fetal Causes

1. Prematurity: It is the commonest cause and accounts for 50% of cases, due to relative excess of the liquor amnii allowing free fetal movement, the center of gravity of the preterm fetus is near its caudal end, also the head of the preterm infant is relatively larger than the breech and so it occupies the roomy fundus.
2. Extension of the legs: This prevents the kicking movement of the fetal legs that helps spontaneous version.
3. Intrauterine fetal death: No kicking movement.
4. Hydrocephalus. The large head occupies the wide fundus.
5. Multiple pregnancy: One fetus prevents turning of the other.

6. Chromosomal anomalies which affect fetal tone and movement as Down syndrome and myotonic dystrophy.

B) Maternal Causes

1. Polyhydramnios: This allows free fetal movement.
2. Oligohydramnios; interfering with spontaneous version.
3. Malformation of the uterus diminishing the capacity of the fundus as septate, sub-septate and bicornuate uterus. These anomalies can lead to recurrent breech presentation.
4. Fundal myoma; diminishing the capacity of the fundus.
5. Multiparity: Lax abdominal and uterine wall allowing free fetal movement.
6. Placenta praevia due to prematurity (preterm labour).

C) Idiopathic

In about 20% of cases, no cause is found.

Notes:

- Contracted pelvis is not a factor in breech presentation.
- There is a higher incidence of congenital fetal anomalies in breech presentations; about 5-10%.

Diagnosis of Complete Breech

I. During Pregnancy

The patient may complain of a hard tender mass in one hypochondrium.

A) Abdominal Examination

1. Inspection

A transverse groove may be seen above the umbilicus if the back is posterior (groove of the neck).

2. Palpation

- a) Fundal level: It corresponds to the period of amenorrhoea.
- b) Fundal grip: The head is felt occupying the fundus.
- c) Umbilical grip: It reveals the position of the back and the limbs.
- d) First pelvic grip: The breech is felt. It is usually not engaged because of its large size.

3. Auscultation

The fetal heart sounds are heard above the umbilicus on the side of the back.

B) Vaginal Examination

In case of doubt a vaginal examination is performed during pregnancy. The soft irregular breech can be differentiated from the hard regular head.

C) Sonography

It confirms the diagnosis, shows the type of breech, diagnoses hyperextended head, estimates gestational age and fetal weight, excludes fetal and uterine anomalies, and diagnoses unsuspected twins.

D) X-ray Examination

X-ray is used to exclude contracted pelvis as other informations are obtained by sonography. Computed tomography is more accurate, gives less radiation, and easier to perform than X-ray pelvimetry. Magnetic resonance imaging is the most accurate method to evaluate the pelvis, and without the risk of radiation, but it is costly, and not available in all centers.

II. *During Labour*

A) Abdominal examination; as before.

B) Vaginal examination; this will reveal

1. Slow dilatation of the cervix, a voluminous bag of forewaters, liability to premature rupture of membranes and prolapse of cord.
2. When the cervix is sufficiently dilated especially after rupture of the membranes, the bony landmarks of the breech are felt; two ischial tuberosities and the sacrum. The feet are felt beside the buttocks. Meconium may stain the examining fingers.

Diagnosis of Breech with Extended Legs

1. Fundal level: The fundus may be at a lower level than the period of amenorrhoea due to early engagement of the breech which is relatively small.
2. Fundal grip: Ballottement of the head is diminished or absent. Small knobs may be felt beside the head.
3. Umbilical grip: The extended limbs may be mistaken for the back.
4. First pelvic grip: The breech feels small, regular and firm and may be mistaken for the head.
5. The fetal heart sounds are heard at or below the umbilicus.
6. Ultrasound may be needed to confirm the diagnosis.
7. During labour frank breech may be mistaken for face presentation.

Mechanism of Labour

1. *Delivery of the Breech*

The bitrochanteric diameter (10 cm) descends in one oblique diameter of the pelvis. The anterior buttock meets the pelvic floor and rotates forwards $1/8$ of a circle. With further descent the anterior buttock appears below the symphysis pubis. By lateral flexion of the spine, the posterior buttock is born at first. By straightening of the spine the anterior buttock is then delivered.

2. *Delivery of the Shoulders*

The biacromial diameter (12 cm) descends in the same oblique diameter as the breech. The anterior shoulder meets the pelvic floor and rotates forwards $1/8$ of a circle. With further descent the anterior shoulder appears below the symphysis pubis and the posterior shoulder is delivered by lateral flexion of the spine followed by the anterior shoulder.

3. *Delivery of the Aftercoming Head*

The head descends in the opposite oblique diameter of the pelvis. The occiput meets the pelvic floor and rotates forwards $1/8$ of a circle in sacro-anterior position and $3/8$ of a circle in sacro-posterior position. With further descent the suboccipital region appears below the symphysis pubis and the head is then delivered in flexion.

Management

I. During Pregnancy

Breech presentation diagnosed at 36 weeks of pregnancy and onwards is corrected by external cephalic version unless there is a contraindication as contracted pelvis.

Advantages of External Cephalic Version

1. To avoid fetal complications associated with breech delivery.
2. To avoid maternal complications associated with breech delivery.
3. To test for cephalopelvic disproportion, especially in the primigravida.

Time

The best time is between 36 and 38 weeks. It is not done before the 36th week because: (1) spontaneous correction usually occurs by the 36th week; (2) the malpresentation is liable to recur; (3) preterm labour may occur due to manipulations. After the 38th week, version is attempted but it usually fails because the volume of the liquor is relatively small in relation to the size of the fetus also the uterus becomes more irritable and sensitive to manipulations. If external cephalic version fails we do X-ray pelvimetry and ultrasonic

examination at 38 weeks to exclude contracted pelvis and to estimate fetal weight to exclude fetopelvic disproportion.

II. During Labour

Delivery must be in hospital. Vaginal delivery is allowed unless there is indication for caesarean section.

First Stage

The same as in normal labour. However, the patient is kept in bed. No walking is allowed to avoid premature rupture of membranes and prolapse of the cord.

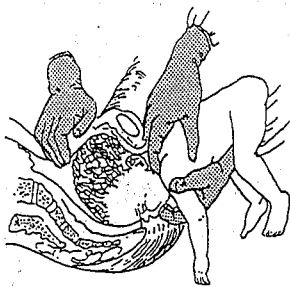
Second Stage

1. When the membranes rupture, vaginal examination is performed to estimate the degree of cervical dilatation, to exclude prolapse of cord and to confirm the diagnosis.
2. The patient is asked to bear down during uterine contractions and to relax in between until the breech appears at the vulva.
3. When the perineum is maximally distended a generous episiotomy is done, this is followed by spontaneous delivery of the breech and trunk up to the umbilicus.
4. The fetus is covered by a warm towel to prevent premature stimulation of respiration. A loop of the cord is drawn down to feel pulsations and to prevent compression of the cord by the fetal head.
5. When the anterior scapula appears below the symphysis pubis, the arms are delivered. This is followed by spontaneous delivery of the shoulders.
6. The aftercoming head is delivered by one of the following methods:
 - a) *Burns-Marshall technique*: The body of the fetus is left hanging down from the edge of the table. The fetus is supported to avoid sudden slipping. The weight of the fetal body applies traction until the suboccipital region appears below the symphysis pubis. The fetus is then moved towards the mother's abdomen to deliver the head in flexion. Suprapubic pressure on the fetal head by an assistant maintains flexion and helps descent of the head (Kristeller method).
 - b) *Jaw Flexion Shoulder Traction (Mauriceau-Smellie-Veit Technique)*: The fetus is put on the left fore-arm with the index and middle fingers introduced in its mouth, or applied over the maxillae to avoid dislocation of the lower jaw. The index and middle fingers of the right hand are applied over the shoulders from behind. Traction is applied downward and backward until the suboccipital region appears below the symphysis pubis then the fetus is moved upward towards the mother's abdomen to deliver the head in flexion.

- c) *Forceps to the aftercoming head:* The forceps is applied from the abdominal aspect of the fetus. Traction is applied downward and backward until the suboccipital region appears below the symphysis pubis. The forceps is then elevated upward to deliver the head in flexion. The forceps should be ready in all breech deliveries. The Piper forceps is preferred because it has a perineal curve on its long shank. If not available a long curved forceps is used. Anaesthesia is necessary for forceps application.

Third Stage

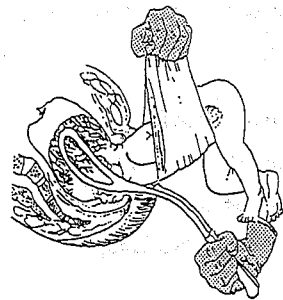
It is managed in the routine way.



Jaw flexion and shoulder traction



Burns-Marshall technique



Applications of Piper forceps

Fig. 77. Methods of delivery of aftercoming head

Indications of Caesarean Section

1. Pelvic contraction (any degree).
2. Placenta praevia (any type).
3. Prolapsed pulsating cord with a partially dilated cervix.
4. Primigravida if elderly or multigravida with no living children (bad obstetric history).
5. Preterm baby with a weight between 1000–1500 gm (28–32 weeks).
6. Large fetus (weight 3500 gm or more, estimated by ultrasound).
7. Footling presentation; as the incidence of cord prolapse is high (15%). Also to avoid head entrapment if the feet descend through a partially dilated cervix.
8. Hyperextended head or "star-gazing" fetus. Diagnosed by X-ray or ultrasound. Such fetus may suffer fracture of the cervical spine during delivery of the head (in 20% of cases). Diagnosed when the angle between the cervical spine and mandible is more than 105° .

9. The presence of other complications as diabetes mellitus, pre-eclampsia and intra-uterine growth restriction. If pregnancy is needed to be terminated in breech presentation, caesarean section is safer for the fetus than induction of labour.
10. Some obstetricians perform caesarean section for all cases of breech presentation particularly in the primigravida to reduce fetal mortality and morbidity but this policy increases the maternal morbidity.

Different Methods of Breech Delivery

1. Spontaneous Breech Delivery

The fetus is born spontaneously by the natural forces without any interference, it is just supported to avoid slipping. It can be applied if the fetus is small, especially in a multipara.

2. Assisted Breech Delivery

The fetus is born spontaneously as far as the umbilicus but we interfere to assist delivery of the shoulders and aftercoming head. It is done in most cases and it is the method described before.

3. Breech Extraction

The entire fetus is delivered by applying traction under anaesthesia. It is indicated if the cervix is fully dilated and there is a need for immediate delivery as fetal distress, maternal distress or prolapsed pulsating umbilical cord. It is also indicated if the second stage is prolonged or to shorten the second stage in maternal diseases as eclampsia and heart failure. Caesarean section is preferable to breech extraction which is dangerous for the fetus.

ABNORMAL (COMPLICATED) BREECH DELIVERY

- I) Arrest of the buttocks.
- II) Arrest of the shoulders.
- III) Arrest of the aftercoming head.

I. Arrest of the Buttocks

A) Arrest of the Buttocks at the Pelvic Inlet

Aetiology

- 1) Contracted pelvis.
- 2) Large fetus.
- 3) Uterine atony.

Management

1. If there is contracted pelvis or big fetus, caesarean section is performed. However, if the fetus is dead it is delivered vaginally by applying traction under anaesthesia. Evisceration and perforation of the head may be needed.
2. Uterine atony: The treatment of inertia including oxytocin drip is given. However, if the arrest persists in spite of good uterine contractions, caesarean section is performed.

B) Arrest of the Buttocks at the Pelvic Outlet (Impacted Breech)

Aetiology

1. Extension of the legs. It is the main cause.
2. Large fetus.
3. Uterine atony.
4. Contracted pelvic outlet.
5. Rigid perineum.

Note: The extended legs interfere with lateral flexion of the spine which is necessary for delivery of the buttocks, also the upper pole of the fetus formed by the head and feet is large and becomes arrested at the pelvic brim.

Management

1. If there is large fetus or contracted pelvic outlet caesarean section is performed.
2. Uterine atony; treated by oxytocin drip or breech extraction.
3. Rigid perineum is treated by episiotomy.
4. Extension of the legs is treated by groin traction or bringing down a leg followed by breech extraction.

Bringing Down a Leg and Breech Extraction (Pinard method)

General anaesthesia, evacuation of the bladder by a catheter and episiotomy. The hand is passed along the anterior thigh to the popliteal fossa. Press by two fingers to flex the leg. The foot is grasped and brought down. If difficult, the hand is passed over the tibia to grasp the ankle and bring down the leg. It is advisable to bring the anterior leg first because if we bring down the posterior leg, the anterior buttock may override the symphysis pubis and labour becomes obstructed. After bringing down both legs breech extraction is carried out.

Groin Traction

May be used when the breech is deeply impacted in the pelvis, so there is no space to introduce the hand. The index finger is put in the anterior groin for traction during uterine contraction, and it may be helped by fundal pressure. When the posterior groin can be

reached, traction is made on both groins. Traction is downward and backward until the anterior buttock appears below the symphysis pubis, and then upward to deliver the posterior buttock. Traction is directed towards the trunk and not towards the thigh, to avoid fracture of the femur. When the fetus is dead a breech hook may be used for traction on the groin.



Fig. 78. Pinard method to bring down the extended legs.

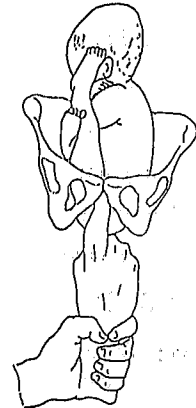
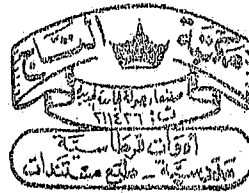


Fig. 79. Groin traction

II. Arrest of the Shoulders

Aetiology

- 1) Extension of one or both arms.
- 2) Nuchal displacement of the arm.



Extension of the Arms

It may be a primary condition but it is usually due to traction on the trunk before the cervix is fully dilated.

Treatment

1. Classical Method

Under general anaesthesia the posterior arm is brought down at first because there is more space in the concavity of the sacrum. The hand is passed along the arm to the cubital fossa. Press by two fingers to flex the forearm. The hand is grasped and pulled down. Now we bring down the anterior arm from behind the symphysis pubis by the same technique. If there is not enough space anteriorly, hold the fetus by its chest and rotate it to bring the anterior arm posteriorly and bring it down by the same procedure. This is followed by delivery of the aftercoming head in the usual way.

2. Lövset Technique

It is the method of choice to bring down the extended arm because it does not need passing the hand into the vagina and it is done without anaesthesia. The fetus is grasped by the pelvic girdle and traction is applied downward until the inferior angle of the anterior scapula appears below the symphysis pubis. Now the fetus is rotated 180° to bring the posterior shoulder below the symphysis pubis where the arm can be brought down. The fetus is then rotated 180° in the opposite direction to bring the other shoulder anterior where the arm can be delivered. The fetus is always rotated in the direction which keeps its back anterior. Downward traction is applied during the whole procedure.

Note: By applying downward traction while the arms are extended the anterior shoulder remains above the symphysis pubis while the posterior shoulder descends to a lower level below the sacral promontory. Now, when we rotate the fetus, the posterior shoulder will appear below the symphysis pubis and the arm can be easily delivered.

Nuchal or Dorsal Displacement of the Arm

The forearm lies behind the back of the neck. The result of rotation of the trunk in the wrong direction in a trial to get down the extended arm. Treated by rotating the fetal trunk in the direction of the tips of the fingers of the displaced hand.

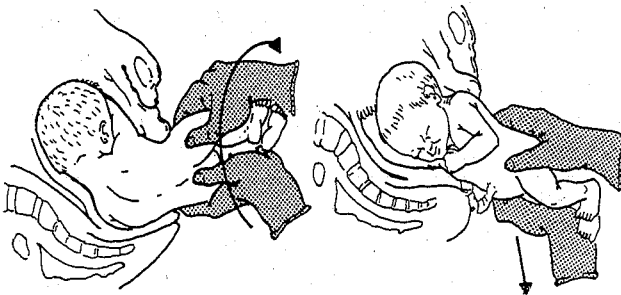


Fig. 80. Lovset technique

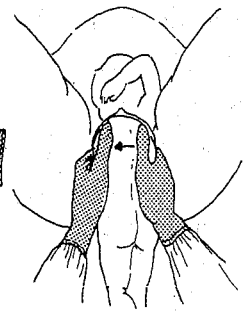


Fig.81. Nuchal position of the arm

III. Arrest of Aftercoming Head

Aetiology

A) Causes in the Head

- 1) Large head.
- 2) Hydrocephalus.
- 3) Extension of the head, bringing a bigger diameter of engagement.

- 4) Posterior rotation of the occiput.

B) Causes in the Passages

- 1) Contracted pelvis.
- 2) Incompletely dilated cervix.
- 3) Rigid perineum.

Management

1. Contracted pelvis or a large head. If the fetus is alive, it is saved by symphysiotomy and if dead craniotomy is performed.
2. Hydrocephalus; treated by craniotomy, or by tapping of the cervical spine (see hydrocephalus).
3. Extension of the head is treated by:
 - a) Burns-Marshall technique, or
 - b) Jaw-flexion and shoulder traction, or
 - c) Application of the forceps.
4. Posterior rotation of the occiput: The fetus is grasped by the chest and rotated to bring the occiput anteriorly. If this fails, Jaw-flexion and shoulder traction is applied to deliver the head as face to pubis. If the mouth is out of reach, the fetus is grasped by one hand from the feet and moved towards the mother's abdomen and the two fingers of the other hand apply downward traction on the shoulders (Prague technique).
5. Rigid perineum is treated by episiotomy.
6. Incompletely dilated cervix: If the fetus is dead, we do craniotomy, if living it can be saved by incisions of the cervix which are sutured immediately after delivery (Dührssen incisions made at 2, 6 and 10 o'clock).

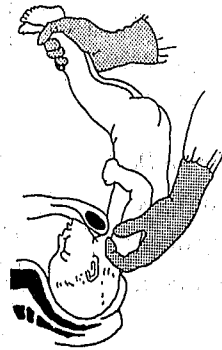


Fig. 82. Prague technique

COMPLICATIONS OF BREECH DELIVERY

I. Fetal Complications

A) *Fatal Injuries*

Fetal mortality in breech delivery is about 3-4% in multigravidae and 6-8% in primigravidae. The causes of death are:

1. *Intracranial haemorrhage*: The commonest cause (50%). It may be traumatic due to rapid compression of the head because there is not enough time for gradual moulding to

occur and due to excessive suprapubic pressure. Fetal asphyxia can also lead to intraventricular haemorrhage particularly in the preterm baby. It can be avoided by:

- a) Slow delivery of the head.
 - b) Avoid rough suprapubic pressure.
 - c) A generous episiotomy to prevent compression of the head by the vulval ring.
 - d) Forceps to the aftercoming head.
 - e) Vitamin K₁ is given to the mother early in labour (10 mg IM) and to the baby immediately after delivery (1 mg IM).
2. *Fracture dislocation of the cervical spine.* The fetus should not be moved towards the mother's abdomen until the suboccipital region appears below the symphysis pubis. Also the fetus should not be moved more than 90°.
3. *Asphyxia* due to:
- a) Premature stimulation of respiration due to exposure of fetal body to the cold atmosphere so the fetus breathes and inhales amniotic fluid. Avoided by covering the body of the fetus by a warm towel.
 - b) Compression of the umbilical cord by the head.
 - c) Premature separation of the placenta due to drop in the intrauterine pressure after delivery of the fetal trunk.
 - d) Retained aftercoming head more than 12–17 minutes. The fetus dies when anoxia lasts more than 12–17 minutes.
4. *Rupture of abdominal organs* as liver or spleen. Avoided by holding the fetus from the hip bones or the chest and not from the abdomen.

B) Non-fatal Injuries

As fracture of clavicle or humerus, dislocation of the lower jaw, brachial plexus palsy and rupture of sternomastoid muscle.

Note: Anoxia (total hypoxia) does not cause any harm to the fetus if it lasts less than 6 minutes. Anoxia lasting 6–12 minutes leads to brainstem necrosis with motor and behavioral changes. Anoxia lasting more than 12–17 minutes leads to fetal death.

II. Maternal Complications

1) Premature rupture of membranes and prolapse of cord; (2) prolonged labour and maternal exhaustion; (3) obstructed labour; (4) uterine atony; (5) postpartum haemorrhage; (6) liability to puerperal sepsis due to prolonged labour, premature rupture of membranes and increased incidence of interference; (7) perineal lacerations because there is not sufficient time for gradual stretching of the perineum to occur, also the long occipito-frontal diameter (11.5 cm) passes through the vulval ring; (8) cervical lacerations if traction is applied on the fetus before the cervix is fully dilated.

EXTERNAL CEPHALIC VERSION

Indications

1. Breech presentation diagnosed at 36 weeks of pregnancy and onwards.
2. Transverse or oblique lie diagnosed at 36 weeks and onwards or early in labour before rupture of membranes.

Time: see page (177).

Technique

1. Ultrasound examination is done to confirm the diagnosis and to exclude fetal or uterine abnormalities that would contraindicate version.
2. The urinary bladder should be empty.
3. Version is done without anaesthesia or analgesia to avoid the use of excessive force.
4. A tocolytic drug as ritodrine (Yutopar) or magnesium sulphate may be given intravenously to relax the uterus if it is irritable. The dose of ritodrine is 150 micrograms/minute IV for 15 minutes or until the uterus is relaxed.
5. The patient lies in a moderate Trendelenburg position (30°) with the hips and knees slightly flexed. The abdomen and vulva are kept uncovered.
6. The position of the fetus is determined and fetal heart sounds (FHS) are auscultated.
7. One hand is applied over the head, the other on the breech. The two poles are pressed together to increase flexion. The head is pushed towards the pelvis, and the breech towards the fundus. The head is pushed in the direction which maintains flexion of the fetus, i.e. moved towards the fetal limbs and away from the back. The manipulations are intermittent. The attendant's hands should be warm.
8. After version is completed, the head is pushed downward and backward into the pelvis and kept in its new position for a few minutes (5 minutes).

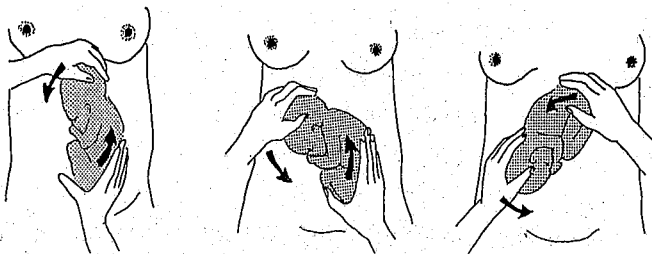


Fig. 83. External cephalic version

9. The FHS are auscultated. If they show persistent fetal distress (more than 5 minutes) the fetus is returned back to its original position because the cord may be coiled. Manipulations are also stopped if vaginal bleeding occurs.
10. The patient is examined after one week. If the breech has recurred the process is repeated.

Causes of Failure

The failure rate is about 5% in multigravidae and 25% in primigravidae. The causes are:

1. Extension of the legs, the commonest cause. The extended legs interfere with complete flexion.
2. Large fetus.
3. Deep engagement of the breech.
4. Undiagnosed twins.
5. Short cord.
6. Scanty or excessive liquor amnii.
7. Irritable uterus.
8. Uterine anomalies as septate uterus.
9. Rigid abdominal wall.
10. Obesity.

Complications

1. Premature rupture of membranes.
2. Preterm labour.
3. Premature separation of the placenta and accidental haemorrhage (abruptio placentae).
4. Knots of the cord or looping of the cord around the fetus.
5. Cord presentation and subsequent prolapse.
6. Fetal shock and death.
7. Rupture of uterus.

Fetal mortality is about 0.3%.

Contraindications

1. Elderly primigravida or multigravida with no living children (bad obstetric history).
2. Severe degree of contracted pelvis.
3. Antepartum haemorrhage (placenta praevia or abruptio placentae).
4. Hypertension for fear of placental separation and haemorrhage (placental abruption).
5. Scar in the uterus, as rupture may occur.
6. Multiple pregnancy.
7. Hydrocephalus.
8. Polyhydramnios. The malpresentation will recur.

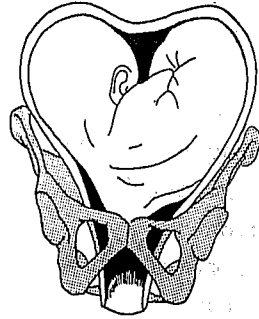
9. If the membranes are ruptured.
10. Before the 36th week.
11. The presence of any indication for caesarean section.

Note: If the mother is Rh-negative and not sensitized, she is given anti-D serum (300 micrograms IM) to prevent sensitization.

SHOULDER PRESENTATION (OBLIQUE OR TRANSVERSE LIE)

Definition

The long axis of the fetus crosses that of the mother with the head on one side and the breech on the opposite side of the middle line.



Incidence

About 1 in 200 labours (0.5%).

Positions

The denominator is the scapula. There are four positions:

Fig. 84. Shoulder presentation

- *Left scapulo-anterior*; back anterior and head to the left.
- *Right scapulo-anterior*; back anterior and head to the right.
- *Right scapulo-posterior*; back posterior and head to the right.
- *Left scapulo-posterior*; back posterior and head to the left.

The scapulo- or dorso-anterior positions are commoner than dorso-posterior positions, because in the former the concavity of the front of the fetus fits into the convexity of the maternal lumbar spine.

Aetiology

I. Maternal Causes

1) Pelvic contraction; (2) pelvic tumour; (3) placenta praevia; (4) polyhydramnios; (5) multiparity with lax abdominal and uterine wall; (6) marked lateral obliquity of the uterus; (7) fundal myoma; (8) insertion of the placenta in the fundus; (9) uterine malformation as bicornuate or subseptate uterus.

II. Fetal Causes

1) Prematurity; (2) intrauterine fetal death (fetus cannot correct itself); (3) multiple pregnancy; (4) short umbilical cord.

Note:

- The commonest cause of shoulder presentation is multiparity.
- Transverse lie recurring in subsequent pregnancies may be due to uterine malformation.

Diagnosis**I. During Pregnancy****A) Abdominal Examination****1. Inspection**

The abdomen looks broader from side to side.

2. Palpation

- a) Fundal level: It is lower than the period of amenorrhoea.
- b) Fundal grip: No head or breech is felt in the fundus.
- c) The umbilical grip: The head is felt on one side and the breech on the other side. They are felt at the same level in transverse lie or one at a lower level than the other in oblique lie. Usually the head is lower because it is heavier.
- d) First pelvic grip: No head or breech is felt.

3. Auscultation

The FHS are best heard on the side of the head.

B) Sonography

This confirms the diagnosis.

II. During Labour**A) Abdominal examination; as before.****B) Vaginal examination, this will reveal**

1. Slow dilatation of the cervix; a voluminous bag of forewaters, liability to premature rupture of membranes and prolapse of cord, arm or both.
2. When the cervix is sufficiently dilated especially after rupture of the membranes the bony landmarks of the shoulder are felt, i.e. the acromion, spine of the scapula and the clavicle, we may also feel the ribs, axilla and the arm. If there is a prolapsed arm the dorsum of the supinated hand will indicate the position of the back, while the thumb will point towards the head of the fetus. The obstetrician can know which arm is prolapsed by shaking hand.

Note: If the shoulder has become oedematous, it may be mistaken for a breech.

Mechanism of Labour

There is no mechanism and labour becomes obstructed. Rarely, if the fetus is small and dead it may be born by spontaneous expulsion or spontaneous evolution.

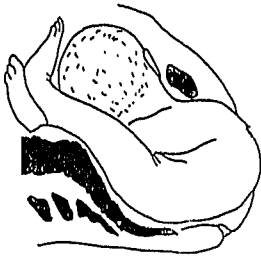


Fig. 85. Spontaneous expulsior



Fig.86. Spontaneous evolution

Spontaneous Expulsion: The fetus becomes compressed and folded like the letter V. The trunk is born at first followed by delivery of the limbs and the head together (Partus Conduplicato Corpore).

Spontaneous Evolution: Overstretching of the neck occurs followed by delivery of the shoulders, trunk then the legs and finally the head.

However, *spontaneous rectification* — i.e. shoulder presentation is changed into cephalic presentation — or *spontaneous version* — i.e. shoulder presentation is changed into breech presentation — may occur during pregnancy or labour and this is followed by delivery as cephalic or breech.

Management

I. During Pregnancy

If the condition is diagnosed after the 36th week of pregnancy and onwards, it is corrected by external cephalic version unless there is a contraindication as contracted pelvis.

II. During Labour.

1. Before Rupture of the Membranes

External cephalic version is performed followed by rupture of the membranes to allow descent of the head in the pelvis. If external version fails, caesarean section is performed.

2. After Rupture of the Membranes

Caesarean section is performed.

Neglected Shoulder

It is a shoulder presentation and the patient was neglected for a long time in labour.

Diagnosis

It is the picture of obstructed labour.

1. *History*: There is a history of prolonged labour with strong and frequent uterine contractions. The membranes are ruptured and the amniotic fluid is drained.
2. *General examination*: This shows evidence of maternal distress as rapid pulse and respiration, high temperature (38°C or above), excessive sweating and signs of dehydration as dry tongue.
3. *Abdominal examination*: The uterus is hard and tender. It is moulded over the fetus. The fetal parts cannot be felt and the FHS are absent. A retraction ring is seen and felt as an oblique groove reaching the umbilicus or even higher.
4. *Vaginal examination*: There is oedema of the vulva. The vagina is warm and dry. The cervix is either fully dilated or it is partially dilated, thick oedematous and hanging. The shoulder is felt impacted in the pelvis, and an arm or cord may be prolapsed.
5. *The urine* is blood-stained as the impacted presenting part leads to bruising of the bladder and rupture of the small capillaries.

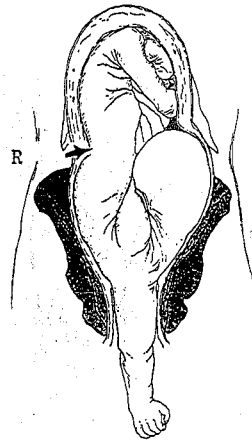


Fig. 87. Neglected shoulder.
R. Pathological retraction ring

Treatment

Caesarean section is performed.

CORD PRESENTATION AND CORD PROLAPSE

Cord presentation means the umbilical cord is found below the presenting part before rupture of the membranes.

Cord prolapse means the umbilical cord is found below the presenting part after rupture of the membranes.

If the cord lies beside but not below the presenting part it is called "occult cord prolapse" which may or may not be felt by vaginal examination.

Incidence

About 1 in 250 labours.

Aetiology

Any factor which interferes with fitting of the presenting part to the lower uterine segment or prevents engagement of the head.

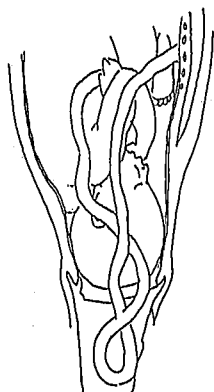


Fig. 88. Prolapse of cord

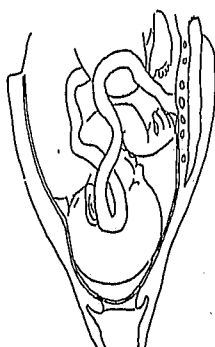


Fig. 89. Occult cord prolapse

I. Fetal Causes

- a) Malpresentation as breech or transverse lie.
- b) Malposition of the head as occipito-posterior, face or brow.
- c) Small head as in case of twins and preterm labour.
- d) Long cord.

II. Maternal Causes

- a) Pelvic contraction, especially flat or oblique pelvis.
- b) Pelvic tumour.
- c) Placenta praevia.
- d) Polyhydramnios.

III. The cord may prolapse after premature or artificial rupture of membranes. External cephalic version may lead to cord presentation and subsequent cord prolapse.

N.B.: The commonest causes are malpresentation and contracted pelvis.

Diagnosis

The condition is expected in any malpresentation or malposition of the head. Vaginal examination should be done in every case after rupture of membranes to exclude cord

prolapse. The prolapsed cord is felt between two fingers to detect pulsations in between uterine contractions. The pulsations may stop completely during a uterine contraction. Occult cord prolapse is rarely diagnosed during vaginal examination. It is suspected if continuous fetal monitoring shows variable decelerations in fetal heart rate. A presenting cord can be felt through the intact bag of membranes, and the pulsations of its vessels should be recognized.

Management

Cord Presentation

A) When the cervix is partially dilated

Caesarean section is performed.

B) When the cervix is fully dilated

The fetus is delivered immediately:

1. If cephalic presentation and the head is not engaged, caesarean section is performed.
2. If cephalic and the head is engaged, deliver by forceps after rupture of membranes.
3. If breech presentation, caesarean section is done. It is preferable to breech extraction which is dangerous for the fetus.

Cord Prolapse

1. If the fetus is dead (non-pulsating cord and FHS are inaudible), we wait for spontaneous delivery. However, if labour is obstructed we interfere according to the type of obstruction, e.g. caesarean section for neglected shoulder.
2. If the fetus is alive, that is prolapsed pulsating cord the treatment depends upon the condition of the cervix:

a) *If the Cervix is Partially Dilated*

Immediate caesarean section, the patient is put in Trendelenburg or knee-chest position, the assistant puts 2 or more fingers in the vagina to push up the presenting part to prevent compression of the cord, and oxygen is given until the theatre is prepared.

b) *If the Cervix is Fully Dilated*

Immediate delivery as mentioned with cord presentation.

Prognosis

As long as the membranes are intact there is no danger to the fetus. After rupture of membranes fetal asphyxia occurs when the cord becomes compressed between the

presenting part and the birth canal or when spasm of the umbilical vessels occurs due to exposure to cold air. Fetal mortality about 20%.

N.B.: *Cord expression:* It means prolapse of the cord with a fully dilated cervix.

COMPOUND (COMPLEX) PRESENTATION

Definition

It is a cephalic presentation in which a limb is found beside the head, usually the hand. Rare, about 1 in 1200 labours.

Aetiology

It is due to factors which prevent fitting of the head against the lower uterine segment. These include:

1. Pelvic contraction, especially flat or oblique pelvis.
2. Small head, as in case of twins, and preterm labour.
3. Sudden escape of liquor amnii in polyhydramnios.

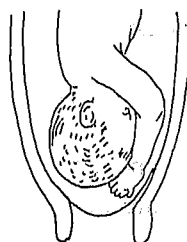


Fig. 90. Compound presentation

Diagnosis

It is diagnosed by vaginal examination during labour when a limb is found beside the head. A prolapsed cord must be excluded because both have the same aetiology.

Management

First Stage of Labour

We wait for spontaneous delivery as the limb usually moves upward into the lower uterine segment as the head descends. However, caesarean section is done if there is contracted pelvis or the patient is elderly primigravida.

Second Stage of Labour

1. *Spontaneous delivery.* If the fetus is small the prolapsed limb may not interfere with spontaneous delivery.
2. *Manual reposition and delivery by forceps under anaesthesia.* If the head is engaged and labour obstructed. The vacuum extractor can be used alternative to forceps.
3. *Caesarean section.* If the head is not engaged and labour obstructed.
4. *Craniotomy.* If the fetus is dead and labour obstructed. However, caesarean section is safer for the mother.

Prognosis

The fetal mortality is about 20% due to prematurity, associated cord prolapse and instrumental delivery.

UNSTABLE LIE

It is frequent change in the fetal lie during the last month of pregnancy. The fetus changes its lie frequently from longitudinal, to oblique, to transverse lie and so on.

Aetiology

(1) Multiparity with lax abdominal and uterine wall allowing free fetal movement (commonest cause), (2) pelvic contraction, (3) pelvic tumour, (4) placenta praevia, (5) polyhydramnios.

Management

The patient is examined at 38 weeks of pregnancy. If the fetal lie is longitudinal induce labour. If the cervix is ripe, induce labour with oxytocin infusion and rupture of the membranes. If the cervix is unripe, induce labour with prostaglandin vaginal tablet or gel. If the fetal lie is oblique or transverse, carry out external cephalic version and then induce labour. If version fails, caesarean section is performed. The aim of interference is to avoid spontaneous onset of labour while the fetus is in an oblique or transverse lie. This leads to obstructed labour and possible uterine rupture before the patient reaches the hospital.

MULTIPLE (MULTIFETAL) PREGNANCY

INCIDENCE

According to Hellin rule the incidence of twins is one in 80 pregnancies, triplets one in 80², quadruplets one in 80³ and so on. This rule applies to spontaneously occurring multiple pregnancies and not those induced by ovulatory drugs as Clomid.

TYPES OF TWINS

Uniovular (Monozygotic, Identical) Twins — 30%

They result from division of a single fertilized ovum. According to the stage of development at which division occurs the uniovular twins may be:

- Dichorionic diamniotic*: 2 placentas, 2 chorions and 2 amniotic sacs. The 2 placentas may fuse together giving rise to a single placenta (division occurs at the morula stage within 3 days after fertilization — 23%)
- Monochorionic diamniotic*: One placenta, one chorion and 2 amniotic sacs (division occurs at the blastocyst stage 4–7 days after fertilization — 75%)
- Monochorionic monoamniotic*: One placenta, one chorion and one amniotic sac (division occurs 8–13 days after fertilization — 1%)
- Conjoined twins*: Due to incomplete division of the fertilized ovum (occurring after 13 days of fertilization). There is one placenta, one chorion and one amniotic sac.

Twins with one amniotic sac have a high fetal mortality (up to 50%) due to entanglement of the cord.

The uniovular twins are identical. They are of the same sex, blood group and appearance but with different finger prints, iris pattern, and voice.

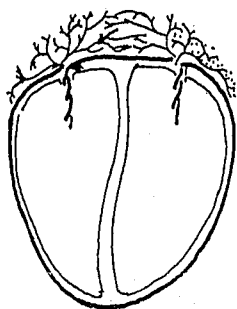


Fig. 91. Uniovular twins

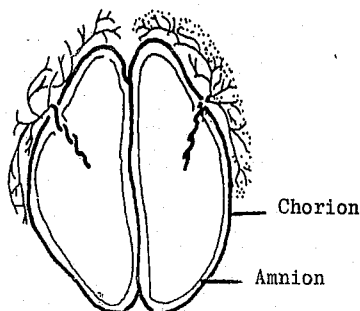


Fig. 92. Binovular twins



Fig. 93. Conjoined twins

Binovular (Dizygotic, Fraternal) Twins — 70%

They result from fertilization of two separate ova. This multiple ovulation is affected by race and familial tendency and is increased with increased maternal age, parity, height, weight (obesity) and drugs which stimulate ovulation as clomiphene and gonadotrophins. The twins are not identical, they may or may not be of the same sex. The similarity between them is like that between two members of the same family. There are 2 placentas, 2 chorions and 2 amniotic sacs (dichorionic diamniotic). However, if the ova are implanted close to each other we will have a single placenta, 2 chorions and 2 amniotic sacs.

The Twin-to-Twin Transfusion Syndrome

The syndrome occurs in about 15% of uniovular twins. Frequently, there is anastomosis between the 2 fetal circulations in the single placenta. This may be artery to artery, artery to vein or vein to vein. When there is artery to vein anastomosis the donor twin becomes hypotensive, anaemic, undergrown with oligohydramnios. The recipient twin becomes hypertensive, polycythaemic, overgrown, with enlarged heart and kidneys leading to increased urination and polyhydramnios. The donor fetus may die from heart failure due to anaemia and the recipient twin may die from congestive heart failure due to over-perfusion. It is usually the recipient fetus which dies. The dead fetus becomes compressed against the uterine wall (fetus compressus or papyraceous). Fetal death occurs in about 20% of cases. The placental vascular anastomosis is diagnosed by Doppler ultrasound.

Superfecundation

It means that two ova produced during the same menstrual cycle are fertilized by two sperms resulting from two separate acts of coitus. This can occur in the human female as shown by the birth of twins, one white and one black, due to successive acts of coitus with a white man and a Negro.

Superfetation

It is the fertilization of two ova produced from two different cycles. This cannot occur in the human female because ovulation is inhibited once pregnancy occurs.

Double Monster

It is a fetus resulting from a single ovum with duplication of head, trunk, or limbs.

PRESENTATION IN TWIN PREGNANCY

The most common presentation is cephalic-cephalic, i.e. both fetuses present by the head (45%). The least common presentation is transverse-transverse (0.5%).

DIAGNOSIS

I. History

There may be a past or a family history of twins (in the family of the wife, husband or both). History of drugs which induce ovulation as Clomid.

II. General Examination

Manifestations of pre-eclampsia are usually present (25–50%).

III. Abdominal Examination

1. Inspection

The abdomen is abnormally huge and overdistended, due to the presence of two fetuses, and due to polyhydramnios which is frequently present.

2. Palpation

The fundal level is higher than the period of amenorrhoea. Palpation shows multiple small parts, the palpation of 3 large poles is a sure sign of twin pregnancy. Sometimes one head is deeply engaged, and so it is felt by the second pelvic grip or by vaginal examination.

3. Auscultation

The auscultation of two fetal heart sounds at two points far away from each other and heard simultaneously by two observers with a difference of at least 10 beats per minute is diagnostic. Arnoux sign is suggestive and not diagnostic and means overlapping of two fetal heart sounds giving a galloping rhythm.

IV. Sonography

This confirms the diagnosis and shows the position and presentation. By sonar the pregnancy sacs appear as white rings as early as 4 weeks by transvaginal ultrasound. At the 12th week, the 2 heads can be detected by sonar.

V. Vaginal Examination

The condition is suspected during labour if the presenting part is small in relation to the size of the uterus.

Note: Twins should be suspected in every case of polyhydramnios.

DIFFERENTIAL DIAGNOSIS

All cases of an oversized pregnant uterus (page 33).

MANAGEMENT

I. During Pregnancy

1. Diet: The mother is given more kilocalories, proteins, iron, calcium and vitamins. Folic acid is given to protect against megaloblastic anaemia (1 mg/day).
2. The patient is given more periods of rest, and warned from liability to preterm labour.
3. The patient is seen more frequent, every 2 weeks starting from 20 weeks till 34 weeks then seen weekly.
4. Serial ultrasound scan (every month) to monitor growth of fetuses. This is started at 20 weeks of pregnancy.
5. Nonstress test is done twice weekly in case of discordant growth. Some do it for all twins as it is a high risk pregnancy.

Notes:

- Admission to hospital between 30 to 34 weeks to prevent preterm labour is no more done as it has no value in this respect.
- Cervical cerclage does not improve the outcome in twin pregnancy, but it is done with triplets and quadruplets.

II. During Labour

1. Delivery must be in hospital.
2. If labour starts before 38 weeks, i.e. preterm, the mother is given 2 g ampicillin IV every 6 hours until delivery, to prevent group B streptococcal infection in the newborn.

First Stage

If the first fetus is vertex, vaginal delivery is allowed and the first stage is managed as in normal labour.

Second Stage

1. When the membranes rupture vaginal examination is done to estimate the degree of cervical dilatation, to exclude prolapse of cord and to confirm the diagnosis of the presenting part.
2. After delivery of the first fetus the cord is immediately clamped and divided to avoid bleeding from the second fetus in case of uniovular twins.
3. The lie of the second fetus is determined. Ultrasound may be needed to determine the lie. If it is longitudinal the second sac is ruptured immediately and delivery is left to

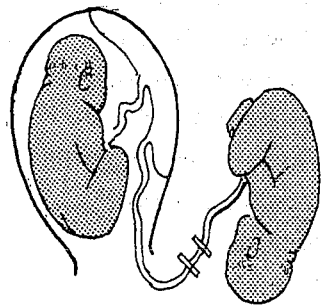


Fig.94. Immediate clamping of the cord

proceed naturally. The fetal heart is monitored continuously. If delivery of the second twin is delayed more than twenty minutes it is extracted by breech extraction if breech, by ventouse or forceps if cephalic and head is engaged or by internal podalic version and breech extraction if cephalic and head is high. If the second twin is oblique or transverse the lie is made longitudinal by external cephalic or external podalic version then the membranes are ruptured or it is delivered by internal podalic version and breech extraction.

4. The patient is kept under observation for 2 hours after delivery to detect any post-partum haemorrhage.

Indications for Caesarean Section

1. If the first fetus is non-vertex.
2. Intrauterine growth restriction (of one or both twins), and twin-to-twin transfusion syndrome.
3. Conjoined twins, and some cases of locked twins.
4. Retained second twin when the cervix reforms and becomes incompletely dilated.
5. If induction of labour is indicated as in case of pre-eclampsia.
6. Any degree of placenta praevia.

COMPLICATIONS

I. Maternal

A) During Pregnancy

1. Hyperemesis gravidarum.
2. Abortion. Sometimes only one fetus is aborted early in pregnancy (vanishing or failed twin). The condition is diagnosed by ultrasound.
3. Preterm labour. Labour occurs in about 50% of twins before 38 weeks.
4. Premature rupture of membranes (25%).
5. Pre-eclampsia (25–50%).
6. Placenta praevia due to large placental site.
7. Increased incidence of pyelonephritis due to marked pressure on ureters.
8. Malpresentation.
9. Nonengagement of the presenting part.
10. Polyhydramnios (10%).
11. Nutritional deficiency and anaemia (iron deficiency and megaloblastic).
12. Pressure symptoms as dyspnoea, palpitation and oedema of the lower limbs.
13. Disseminated intravascular coagulation (if a dead twin is retained. It is due to release of thromboplastin from the dead fetus).

B) During Labour

1. Premature rupture of membranes.
2. Prolapse of arm, cord or both.
3. Obstructed labour. The causes are: first twin is transverse, conjoined or locked twins, and double-headed monster.
4. Abruptio placentae due to premature separation of the placenta due to drop of intra-uterine pressure after delivery of the first twin.
5. Uterine atony due to overdistension of the uterus.
6. Retained placenta due to uterine atony.
7. Postpartum haemorrhage due to atony and large placental site.
8. Splanchnic shock. The marked pressure of the uterus on the splanchnic vessels drops after delivery leading to pooling of blood in the splanchnic area and shock.
9. Locking of twins. A very rare complication

C) During Puerperium

1. The uterus may take a longer time to involute (subinvolution).
2. Puerperal sepsis due to premature rupture of membranes and manipulations.

II. Fetal

1. Prematurity and its complications as respiratory distress syndrome, and infection.
2. Intrauterine growth restriction due to placental insufficiency, or transfusion syndrome. It may affect one or both fetuses. Sometimes one fetus dies and becomes compressed against the uterine wall (fetus compressus or papyraceous).
3. Twin-to-twin transfusion syndrome. This may lead to death of one fetus usually the recipient twin.
4. Increased incidence of congenital fetal malformation (nearly double the incidence in singleton pregnancies).

LOCKING OF TWINS

It occurs when the first fetus is breech and the second fetus is cephalic or transverse. The chin of the first baby locks in the neck of the second. It is a very rare complication, occurs about 1 in 1000 twin deliveries, i.e. 1 in 80,000 of all deliveries. Locking leads to obstructed labour. It is treated by trying unlocking under general anaesthesia. If this fails the first fetus is delivered by decapitation. This is followed by



Fig.95. Locked twins

delivery of the second fetus by internal podalic version and breech extraction. Finally the head of the first fetus is extracted. Some advise caesarean section to deliver the second twin from above.

TRIPLET PREGNANCY

Pregnancy with 3 or more fetuses (multifetal pregnancy) is better treated by caesarean section unless the fetuses are markedly premature.

SELECTIVE EMBRYO REDUCTION

It is done in the first trimester, in the presence of 3 or more fetuses. The number is usually reduced to a twin pregnancy to reduce the fetal morbidity and mortality resulting from preterm labour. Under general anaesthesia, a needle is passed abdominally or vaginally under ultrasound guide to inject air, hypertonic saline or a small amount of potassium chloride into the fetal thorax or heart (about 1.5 ml of 15% solution). This causes cardiac arrest and death of the embryo. Also, the needle can be passed transcervically to aspirate the embryo by repeated suction. Potassium chloride may diffuse and affects other fetuses.

Notes for the postgraduate

1. Ultrasound diagnosis of discordant fetal growth. Ultrasound shows a difference in (1) biparietal diameter ≥ 5 mm; (2) head circumference $\geq 5\%$; (3) abdominal circumference ≥ 20 mm; (4) fetal weight $\geq 20\%$. The last 2 findings are more reliable. Discordant growth is explained by unequal placental mass, twin-to-twin transfusion syndrome, or genetic cause.
2. Treatment of twin-to-twin transfusion syndrome. See fetal therapy.

POLYHYDRAMNIOS (HYDRAMNIOS)

DEFINITION

It means excessive amniotic fluid which is detected clinically or by ultrasound. In this case the amount of fluid is more than 2 litres. By ultrasound the vertical diameter of the largest pocket of amniotic fluid measures 8 cm or more, or the amniotic fluid index (AFI) is 20 cm or more.

AETIOLOGY

The amniotic fluid has both fetal and maternal origin, so the causes of polyhydramnios are:

A) Fetal Causes

1. Twins. Uniovular or binovular. Acute polyhydramnios is almost always associated with uniovular twins. Polyhydramnios affects the sac of the larger fetus due to hypertrophy of its kidneys with increased urination.
2. Congenital fetal anomalies (25%):
 - a) Anencephaly and open spina bifida due to loss of cerebrospinal fluid from the exposed meninges. Other factors in anencephaly are irritation of the uncovered cerebrospinal centers with increased urination, lack of antidiuretic hormone and impairment of swallowing may also play a role.
 - b) Congenital obstruction of the oesophagus or duodenum (interfering with swallowing of amniotic fluid).
 - c) Hypoplastic lungs (interfere with inspiration of amniotic fluid and its absorption by bronchial mucosa and leads to polyhydramnios).
3. Knot of the umbilical cord, cardiac abnormalities, and cirrhosis of fetal liver. These lead to obstruction of circulation in the umbilical vein, oedema of the placenta and increased transudation
4. Chorioangioma which is a benign tumour of the placenta (50% are associated with polyhydramnios). It is a haemangioma of fetal capillaries.
5. A large placenta (leading to increased transudation of amniotic fluid).

Note: The causes of fetal liver cirrhosis are congenital syphilis, erythroblastosis fetalis, cytomegalovirus and toxoplasmosis.

B) Maternal Causes

1. Causes of generalized oedema as heart or renal failure.
2. Pre-eclampsia due to oedema of placenta and membranes.
3. Diabetes mellitus, due to polyuria of the fetus because of hyperglycaemia or increased osmotic pressure of liquor amnii due to the presence of sugar.

4. Syphilis, if it causes cirrhosis of fetal liver.
5. Maternal prolactin may play a role. Prolactin controls the passage of water and solutes across the fetal membranes. In polyhydramnios the level of prolactin in amniotic fluid is lower than that expected for gestational age.

C) Idiopathic

In about 30% of cases no cause is found.

Notes:

- The fetus swallows and inspires amniotic fluid and urinates in the amniotic sac.
- *The amniotic fluid index.* The umbilicus divides the uterus into upper and lower halves, and the linea nigra divides the uterus into right and left halves. The vertical diameter of the largest amniotic fluid pocket in each quadrant is measured in centimeters. AFI is the total of these measurements. AFI 20 cm or more indicates polyhydramnios. AFI 5 cm or less indicates oligohydramnios.

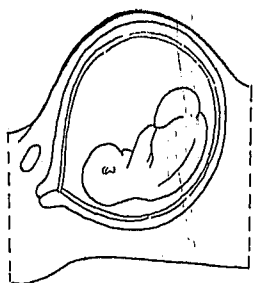


Fig.96. Polyhydramnios

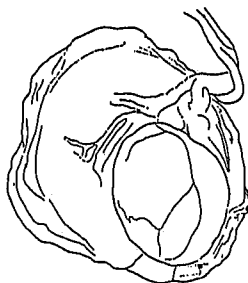


Fig. 97. Chorioangioma of the placenta

CLINICAL PICTURE

I. Acute Polyhydramnios

It is very rare (1 in 3,000 pregnancies), usually occurs in early pregnancy (16–20 weeks) and is almost always associated with uniovular twins. A large amount of fluid accumulates in a few days, it leads to abortion or preterm labour.

A) Symptoms

1. Abdominal pain.
2. Nausea and vomiting.
3. Pressure symptoms: as dyspnoea, palpitation and oedema of lower limbs, vulva and abdominal wall (interference with venous return).

B) Signs

1. *General examination:* Pre-eclampsia is usually present.

2. *Abdominal examination:* The abdomen is abnormally huge and overdistended. The fundal level is higher than the period of amenorrhoea. Fetal parts and fetal heart sounds cannot be detected. There is a fluid thrill.
3. *Vaginal examination:* The cervix is high and usually partially dilated admitting 1 or 2 fingers.

II. Chronic Polyhydramnios

Commoner than the acute, about 1 in 100 singleton pregnancies. Usually occurs in late pregnancy, the fluid accumulates slowly.

A) Symptoms

1. Abdominal discomfort.
2. Pressure symptoms as dyspnoea, palpitation and oedema of the lower limbs, vulva and abdominal wall.

B) Signs

1. *General examination:* Pre-eclampsia is usually present.
2. *Abdominal examination:* The abdomen is abnormally huge and overdistended. The fundal level is higher than the period of amenorrhoea. The fetal parts are difficult to feel or may be felt by dipping or deep palpation, malpresentations are common. The fetal heart sounds are faint or inaudible. There is a fluid thrill.
3. *Vaginal examination:* The cervix is high and usually partially dilated admitting 1 or 2 fingers.
4. *Ultrasound examination:* It must be done in every case of polyhydramnios to confirm the diagnosis, to exclude multiple pregnancy, to show fetal abnormalities, and to diagnose position and presentation.

DIFFERENTIAL DIAGNOSIS

1. Causes of an oversized pregnant uterus.
2. Ascites.
3. Ovarian cyst with pregnancy.

MANAGEMENT

I. Acute Polyhydramnios

Pregnancy is terminated by rupture of membranes (amniotomy). The liquor is allowed to escape slowly by putting 2 fingers in the cervix to avoid splanchnic shock and bleeding from premature separation of the placenta.

II. Chronic Polyhydramnios

A) During Pregnancy

An ultrasound examination should be done. If there is congenital fetal abnormality pregnancy is terminated by amniotomy. If there is no fetal anomaly, the treatment will be:

1. Rest in bed.
2. Glucose tolerance test is done except in mild cases.
3. Treatment of the cause if detected as diabetes mellitus.
4. The prostaglandin inhibitor indomethacin (Indocid). It can be given orally or rectally to avoid gastric irritation. The oral dose is 25 mg every 6 hours or a suppository 100 mg twice daily. It is given for 3 or 4 days but not more to avoid fetal complications, but can be repeated after 5 days. The drug acts on the fetal kidneys reducing the amount of urine and increases the fetal respiratory movements thus increasing the absorption of amniotic fluid by the bronchial mucosa. See tocolytic drugs for fetal complications.
5. Amniocentesis. Indicated if pressure symptoms are severe and not relieved by treatment. Under local anaesthesia, and ultrasound guide, a plastic catheter is passed and amniotic fluid is drained slowly at a rate of 500 ml per hour. About 1500-2000 ml are removed. Procedure may be repeated as the fluid reaccumulates again. Maternal complications include rupture of membranes, preterm labour, chorioamnionitis, placental abruption if a large volume is removed, and bowel injury. Fetal complications include injury to the fetus, umbilical cord, and placenta. Trauma to the placenta can cause maternal isoimmunization and haemolytic disease in the fetus.

B) During Labour

1. Correction of any malpresentation.
2. When the cervix is half dilated the membranes are ruptured and the liquor is allowed to escape slowly by putting 2 fingers in the cervix, to avoid splanchnic shock, bleeding from premature separation of the placenta and prolapse of the cord or arm. To avoid these complications the hindwaters may be ruptured by the Drew-Smythe catheter.
3. The patient is kept under observation for 2 hours after delivery to detect any post-partum haemorrhage.

COMPLICATIONS

I. Maternal

A) During Pregnancy

1. Abortion (as a result of overdistension of the uterus).
2. Preterm labour.

3. Premature rupture of membranes.
4. Pre-eclampsia (present in about 25% of cases).
5. Placenta praevia (due to large placenta).
6. Malpresentation.
7. Nonengagement of the presenting part.
8. Pressure symptoms: as dyspnoea, palpitation and oedema of lower limbs.

B) During Labour

1. Premature rupture of membranes.
2. Prolapse of arm, cord or both.
3. Obstructed labour due to malpresentation.
4. Abruptio placentae due to rapid escape of liquor with premature separation of the placenta.
5. Uterine atony due to overdistension of the uterus.
6. Retained placenta, due to uterine atony.
7. Postpartum haemorrhage due to uterine atony.
8. Splanchnic shock, occurs if the fluid escapes rapidly, so the pressure exerted by the uterus on the splanchnic vessels drops suddenly leading to pooling of blood in the splanchnic area and shock.

C) During Puerperium

The uterus may take a longer time to involute (subinvolution).

II. Fetal

1. Prematurity and its complications as respiratory distress syndrome and infection.
2. Congenital fetal malformations are frequent (about 25%)

CARE OF THE NEWBORN

After delivery the fetus is examined for congenital malformation and a soft rubber catheter is passed into the stomach to test for oesophageal atresia.

OLIGOHYDRAMNIOS

DEFINITION

It means diminished amniotic fluid which is detected clinically or by ultrasound. In this case the amount of the fluid is less than 500 ml. By ultrasound the vertical diameter of the largest pocket of amniotic fluid measures 2 cm or less, or the amniotic fluid index is 5 cm or less.

INCIDENCE

0.5–5% of all pregnancies.

AETIOLOGY**A) Fetal Causes**

1. Congenital anomalies of the fetal urinary tract as bilateral renal agenesis (Potter syndrome), renal dysplasia and posterior urethral valve in a male fetus. There is decreased production or excretion of fetal urine.
2. Fetal chromosomal abnormalities as trisomies.
3. Intrauterine growth restriction. There is decreased production of fetal urine.
4. Postterm pregnancy (placental aging and insufficiency).

B) Maternal Causes

1. Premature rupture of membranes.
2. Placental insufficiency as in pre-eclampsia and chronic hypertension.
3. Diabetic vasculopathy. Atherosclerosis of pelvic arteries which occurs in advanced diabetes (class D and above) reduces uteroplacental blood flow.

C) Idiopathic

No apparent cause is detected.

Notes:

- Indomethacin (Indocid) is a prostaglandin inhibitor which causes oligohydramnios and can be used for treatment of polyhydramnios.
- Potter syndrome is bilateral renal agenesis which is always associated with other deformities as large (bat-like) low-set ears deficient in cartilage, flat nose, with a crease below the lower lip, recession of the chin and joint contractures.

COMPLICATIONS

1. Fetal pulmonary hypoplasia (compression of the thorax by the uterus preventing lung expansion).
2. Club foot is another deformity.
3. Formation of adhesions between uterine wall and fetal skin. This may result in amputation of a limb.
4. Breech presentation is common. Oligohydramnios interferes with spontaneous cephalic version.
5. During labour there is slow dilatation of the cervix due to a small bag of forewaters. Also fetal asphyxia may occur due cord compression.
6. Increased incidence of meconium aspiration syndrome. Fetal asphyxia during labour, which results from cord compression, leads to passage of meconium, as asphyxia

stimulates intestinal movements and relaxes the external anal sphincter. Also the small amount of liquor cannot dilute the meconium. This leads to the presence of thick meconium which is inhaled by the fetus leading to obstruction of the airway and neonatal asphyxia.

DIAGNOSIS

1. The fundal level is lower than the period of amenorrhoea.
2. Breech presentation is common.
3. The fetal parts are easily felt and the fetus is almost immobile. The FHS are clearly heard.
4. Ultrasound scan diagnoses reduced amount of liquor, intrauterine growth restriction, fetal presentation and the presence of fetal anomalies as renal agenesis.

MANAGEMENT

During labour, the fetus is observed for asphyxia. If it occurs the fetus is delivered immediately, either vaginally or by caesarean section according to the condition.

Amniofusion can be done during labour to prevent compression of umbilical cord and fetal asphyxia.

ABNORMAL UTERINE ACTION (UTERINE DYSFUNCTION - UTERINE DYSTOCIA)

CLASSIFICATION

1. Uterine overactivity:
 - a) Precipitate labour.
 - b) Tonic contraction and retraction of the uterus.
2. Uterine underactivity or hypotonic inertia.
3. Uterine irregular activity (incoordinate uterine action or hypertonic inertia).
 - a) Hypertonic lower uterine segment.
 - b) Colicky uterus.
4. Contraction ring.
5. Cervical dystocia.

Note: Dystocia means difficult labour. Inertia means lack of activity.

I. UTERINE OVERACTIVITY

A) Precipitate Labour

The fetus is rapidly expelled from the birth canal. The duration of labour is less than 3 hours (less than one hour by some authorities), sometimes few minutes.

Aetiology

1. Strong frequent uterine contractions.
2. Laxity of the tissues of the birth canal, so more frequent in multipara.
3. High pain threshold, so the patient does not feel except the last few strong contractions.

Complications

A) Maternal

1. Lacerations of the cervix, vagina or perineum.
2. Postpartum haemorrhage (due to lacerations and exhausted uterus as it did much work in a short time).
3. Inversion of uterus.
4. Rupture of symphysis pubis.
5. Acute mania.
6. Puerperal sepsis due to lacerations and unsuitable circumstances.
7. Amniotic fluid embolism.

B) Fetal

1. Asphyxia: The strong frequent uterine contractions interfere with placental circulation.
2. Intracranial haemorrhage due to rapid compression of the head.
3. Rupture of the umbilical cord.
4. Injury or death of the fetus due to falling.

Treatment

If there is a previous history of precipitate labour, the patient is admitted to hospital few days before the expected time of delivery. If the case is seen before delivery of the fetus, inhalation anaesthesia (as nitrous oxide and oxygen) is given to slow the course of labour and episiotomy is performed to avoid perineal tears and intracranial haemorrhage. Tocolytic drug as "Yutopar" may be effective. If the case is seen after delivery, the birth canal is examined for lacerations, and the fetus for injuries.

B) Tonic Contraction and Retraction of the Uterus**"High Retraction Ring – Bandl Ring"****Aetiology**

It is due to obstructed labour. The upper uterine segment becomes markedly contracted and retracted. The lower segment becomes thinned and stretched. The retraction ring rises to a high level and is called the pathological retraction ring or Bandl ring.

Clinical Picture

It is the picture of obstructed labour or impending rupture of uterus.

1. There is a *history* of prolonged labour with strong and frequent uterine contractions. The membranes are ruptured and the amniotic fluid is drained.
2. *General examination* shows maternal distress as rapid pulse and respiration, high temperature (38°C or above) excessive sweating, and signs of dehydration as dry tongue.
3. *Abdominal examination*: The uterus is hard and tender. It is moulded over the fetus. The fetal parts cannot be felt. The FHS are absent or show fetal distress. A retraction ring is seen and felt as an oblique groove reaching the umbilicus or even higher (the obliquity of the line differentiates it from a full bladder).
4. *Vaginal examination*: There is oedema of the vulva, the vagina is warm and dry. The cervix is either fully dilated or partially dilated, thick, oedematous and hanging. The presenting part is high above the brim or impacted in the pelvis. In cephalic presentation there is excessive moulding and a large caput is felt.
5. *The urine* is blood-stained as the impacted presenting part leads to bruising of the bladder and rupture of the small capillaries.

Treatment

It is treatment of the cause of obstruction, e.g. caesarean section for contracted pelvis.

II. HYPOTONIC UTERINE INERTIA

The uterine contractions are weak, infrequent and of short duration. They are not accompanied by marked hardening of the uterus.

Types

1. *Primary hypotonic inertia*: Uterine atony present from the beginning of labour.
2. *Secondary hypotonic inertia*: Uterine atony developing after a period of normal contractions (exhausted uterus).

Aetiology

The condition may be associated with the following:

1. General Causes

Anaemia, antepartum haemorrhage, malnutrition, elderly primigravida, grand multipara, excessive sedation and deep anaesthesia.

2. Local Causes

- Overdistension of the uterus by twins, polyhydramnios, and a large fetus.
- Uterine hypoplasia.
- Fibroids which mechanically interfere with uterine contractions.
- Malposition of the head as face, brow and occipito-posterior.
- Malpresentation as breech and transverse lie.
- Disproportion.

In the last 3 conditions, there is absence of reflex polarity.

3. Nervous Causes

Anxiety and fear. Full bladder or rectum reflexly inhibits uterine contraction.

4. Dystocia Dystrophia Syndrome

The patient is short, fatty, hirsute, plethoric, with android pelvis. The middle 3 fingers are approximately of the same length. She is liable to infertility, abortion, pre-eclampsia and uterine atony during labour.

5. Idiopathic

In many cases, the cause is unknown (about 50%).

Note: Anxiety and fear increase the catecholamines which antagonize the action of oxytocin on myometrium.

Complications

1. Prolonged labour leading to fetal and maternal distress.
2. Failure of internal rotation in occipito-posterior and mento-posterior position.
3. Retention of the placenta.
4. Postpartum haemorrhage.
5. Congenital fetal pneumonia and puerperal sepsis if there is premature rupture of membranes or labour is prolonged more than 24 hours.

Management

A) General Treatment

1. Exclude disproportion and reassure the patient.
2. Keep the bladder and rectum empty.
3. If the patient is dehydrated, concentrated glucose is given I.V.
4. Antibiotics are given if there is premature rupture of membranes more than 18 hours or labour prolonged more than 24 hours.
5. Observation of the mother and fetus.

B) Amniotomy

The membranes are ruptured if still intact provided the head is engaged or at least well applied to the pelvic inlet (to avoid cord prolapse). If the contractions do not improve within one hour after amniotomy, oxytocin infusion is started

C) Oxytocin Treatment

Given unless there is a contraindication as disproportion. Ten units of oxytocin (Syntocinon) in 500 ml of 5% glucose in normal saline given intravenously by the drip method. Begin by 2 milliunit/minute (2 drops per minute) and gradually double the strength every 15 minutes according to the uterine response, but do not exceed 32 mU/min (32 drops/min).

D) Operative Treatment

Indicated if there is fetal or maternal distress or if the cervix fails to dilate for 2 hours (failure of progress). If the cervix is not fully dilated, caesarean section is done. If the cervix is fully dilated apply the forceps or ventouse and if breech do caesarean section. It is preferable to breech extraction which is dangerous for the fetus.

III. HYPERTONIC UTERINE INERTIA

A) Hypertonic Lower Uterine Segment

The contractions in the upper segment are weak while the lower segment is hypertonic resisting dilatation and stretch (reversed polarity). There is severe lower abdominal pain and backache. The condition is often associated with occipito-posterior position giving rise to the so called "posterior position pains".

B) Colicky Uterus

The uterine contractions are irregular and colicky. They are ineffective because dilatation of the cervix occurs slowly and labour is prolonged. The membranes rupture prematurely. Fetal and maternal distress develop rapidly. The patient feels pain even between contractions due to raised intrauterine pressure. The abdomen is tender and the patient usually refuses examination.

Treatment of Hypertonic Inertia

1. General treatment as before.
2. Give an analgesic or antispasmodic. The best is pethidine.
3. Caesarean section if there is fetal or maternal distress or if the cervix fails to dilate for 2 hours.

IV. CONTRACTION "CONSTRICTION" RING

It is a localized spasm of the circular muscle fibres of the uterus. It usually occurs around a groove in the body of the fetus, e.g. the neck but in some cases it is below the presenting part. It may occur in the third stage of labour leading to retention of the placenta and postpartum haemorrhage (hour glass contraction of the uterus). It is usually preceded by a colicky uterus.

Aetiology

The true cause is unknown. The following are predisposing factors:

1. Obstructed labour.
2. Prolonged labour (a fatigue phenomenon).
3. Misuse of uterine stimulants.
4. Repeated intrauterine manipulations under light anaesthesia, such as internal version.
5. Premature rupture of membranes, malpresentation and malposition of the head as occipito-posterior.

Diagnosis

The presence of a contraction ring is suspected in the following conditions:

1. If labour is prolonged in spite of good uterine contractions and absence of obstruction.
2. The presence of continuous pain in the lower abdomen.
3. The presence of colicky uterine contractions.
4. On vaginal examination the head does not descend during contraction but it recedes upwards.

The sure diagnosis is only reached when the cervix is sufficiently dilated to feel the ring.

Treatment

1. If it is suspected in the first stage give an analgesic or antispasmodic. The best is pethidine.
2. During the second stage, the patient is given general anaesthesia and halothane is used to relax the ring and the fetus is immediately delivered by forceps. If the ring fails to relax and the fetus is alive caesarean section is performed. It is a longitudinal incision in the lower segment to divide the ring. If the ring fails to relax and the fetus is dead, continuous traction is made by applying a weight of 2–3 pounds to the forceps until the ring relaxes.
3. In the third stage; retained placenta due to hour glass contraction is treated by giving general anaesthesia and using halothane followed by manual removal of the placenta.

V. CERVICAL DYSTOCIA

It means failure of the cervix to dilate within a reasonable time, i.e., 2 hours, in spite of good uterine contractions. It may be:

1. Primary cervical dystocia (functional rigidity of the cervix) or,
2. Secondary cervical dystocia (organic rigidity of the cervix).

Primary Cervical Dystocia

The cervix appears normal. Failure of dilatation is due to lack of softening of the cervix during pregnancy, cervical hypoplasia or cervical spasm due to overactivity of the sympathetic nervous system (tense mind → tense cervix).

Secondary Cervical Dystocia

It is due to organic lesion of the cervix as benign and malignant tumours or excessive fibrosis occurring after amputation or cauterization of the cervix.

Complications

1. Prolonged labour with fetal and maternal distress.
2. Rupture of uterus.
3. Annular detachment of cervix.

Treatment

- a) Organic rigidity is treated by caesarean section.
- b) The treatment of functional rigidity includes:
 1. General treatment as mentioned before with hypotonic inertia.
 2. Give an analgesic or antispasmodic. The best is pethidine.
 3. Caesarean section if there is fetal or maternal distress or if the cervix fails to dilate for 2 hours (failure of progress).

<i>Retraction Ring</i>	<i>Contraction Ring</i>
1. Occurs at the junction of the upper and lower uterine segments.	1. May occur anywhere in the uterus, usually around a groove in the body of the fetus, e.g. the neck.
2. General examination: There is maternal distress.	2. No maternal distress.
3. Abdominal examination: The uterus is hard and tender. The uterus is moulded over the fetus, the fetal parts cannot be felt. The FHS are absent or show fetal distress. The ring is seen and felt as an oblique groove at or above the level of the umbilicus.	3. The ring is not felt or seen abdominally. The uterus is not tender. The fetal parts are felt. The FHS are normal.
4. Vaginal examination: Signs of obstructed labour.	4. No signs of obstructed labour.
5. If neglected we get rupture of uterus.	5. No danger of rupture of uterus.

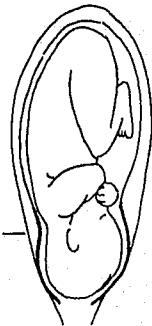


Fig. 99. Contraction ring



Fig. 98. Retraction ring

OBSTRUCTED LABOUR

Definition

Failure of delivery of the fetus in the presence of strong uterine contractions.

Aetiology

I. Fetal Causes

1. Large fetus.
2. Malposition of the head as persistent occipito-posterior, mento-posterior, or brow.
3. Malpresentation as transverse or oblique lie.
4. Locked or conjoined twins.
5. Congenital fetal anomalies as hydrocephalus.

II. Maternal Causes

1. Contracted pelvis.
2. Contraction ring opposite the neck of the fetus.
3. Cervical rigidity organic or functional.
4. Stenosis, septa or tumours of the vagina.
5. Rigid perineum.
6. Pelvic tumour.
7. Pendulous abdomen. The fetus is pushed against the posterior uterine wall and not towards the pelvic inlet.



Clinical Picture (see page 211).

Outcome of Obstructed Labour

1. In the primigravida the uterus responds to obstruction by becoming inert, i.e. secondary hypotonic inertia.
2. In the multigravida the upper uterine segment continues to contract and retract leading to overstretching of the lower uterine segment which finally ruptures.

Complications

A) Fetal

1. Asphyxia and even death due to interference with placental circulation.
2. Infection in the form of congenital pneumonia and septicaemia.
3. Intracranial haemorrhage due to excessive moulding.

B) Maternal

1. Maternal distress.
2. Rupture of uterus.
3. Necrotic fistula.
4. Puerperal sepsis.

Treatment

It is treatment of the cause, e.g. caesarean section for contracted pelvis. When vaginal delivery is difficult or dangerous caesarean section is performed even if the fetus is dead (as in case of neglected shoulder).

OVERSIZED FETUS

It is due to either generalized fetal enlargement, i.e., macrosomia, or localized fetal enlargement. The latter can be due to hydrocephalus, encephalocele, meningocele, hydrothorax, ascites, and tumours.

MACROSOMIA

This means the fetal weight at birth is 4 kg or 4.5 kg (in USA) or more, or the birth weight is above the 90th percentile for gestational age.

Incidence

About 5-10% with all pregnancies, but 25-50% with gestational and pregestational diabetes mellitus.

Aetiology

- 1) Large size of one or both parents, especially the mother (genetic factor).
- 2) Maternal obesity (more than 20% above the ideal body weight). This is the most common predisposing factor.
- 3) Excessive maternal weight gain during pregnancy (>20 kg).
- 4) Maternal diabetes mellitus.
- 5) Multiparity.
- 6) Postterm pregnancy.
- 7) Hydrops fetalis (immune and nonimmune).
- 8) Previous macrosomic baby.
- 9) Fetal gigantism (Beckwith-Wiedmann syndrome).

Complications

- 1) Obstructed labour with increased rate of caesarean section, forceps, and ventouse delivery. Also rupture of uterus may occur.

- 2) Shoulder dystocia, and its complications (fetal and maternal).
- 3) Cervical, vaginal and perineal lacerations and extension of episiotomy.
- 4) Postpartum haemorrhage (PPH) due to lacerations and uterine atony because of over-distension by the large fetus.
- 5) Newborn obesity may predispose to obesity in later life.

Shoulder dystocia and PPH are the most common complications.

Management

- 1) Proper control of hyperglycaemia in diabetic patients.
- 2) Proper control of weight gain during pregnancy.
- 3) During labour, vaginal delivery is allowed, however, caesarean section is indicated in the following conditions:
 - a) Fetal weight 4.5 kg or more.
 - b) Fetal weight 4 kg or more if the mother is diabetic because shoulder dystocia is more common in diabetic patients.
 - c) Past history of shoulder dystocia or Erb's palsy.
 - d) Breech presentation.

Note: The macrosomia of diabetic mother is asymmetric due to increase in both fat and muscle tissue while the brain size is not altered. This leads to increase in the size of the shoulders and abdomen while head circumference is normal. This predisposes to shoulder dystocia. The macrosomic infant of the nondiabetic mother shows increased growth of both head and abdominal circumferences, i.e., symmetric macrosomia. For this reason the abdominal circumference is the most reliable diameter to diagnose macrosomia.

HYDROCEPHALUS

Definition

It is due to excessive accumulation of cerebrospinal fluid (CSF) in the ventricles of the brain leading to enlargement of the head. Associated abnormalities as spina bifida are common (in about 30% of cases). The volume of fluid is usually between 500 and 1500 ml, but may reach 5 litres.

Incidence

About 1 in 2000 fetuses.

Aetiology

It is due to obstruction in the flow, or increased production of CSF. Obstruction of the aqueduct of Sylvius may be due to (a) genetic disorders as trisomies 13, 18, and 21, (b) infection due to toxoplasmosis, rubella, and cytomegalovirus, (c) intracranial tumours, (d) intracerebral haemorrhage.

Diagnosis

During pregnancy, the head is felt large with soft bones. Breech presentation is common (in 30% of cases). Sonography confirms the diagnosis. During labour vaginal examination reveals wide sutures, large fontanelles and thin, soft, indentable cranial bones.

Treatment

Hydrocephalus leads to obstructed labour. The head is perforated vaginally using the perforator or it is tapped vaginally or abdominally using a spinal needle or a trocar (cephalocentesis). This is followed by spontaneous delivery. In breech presentation the aftercoming head is either perforated or spinal tapping is carried out. A metal cannula is passed up through the spinal canal (via a spina bifida if present). Tapping is easier and safer than perforation. In selected cases with mild hydrocephalus and good thickness of cerebral cortex determined by ultrasound (1 cm) caesarean section may be done and the newborn infant will then have a shunt operation. A ventriculo-amniotic shunt with a one way valve may be done with the fetus in utero to drain CSF from cerebral ventricles into the amniotic sac to prevent compression and atrophy of brain tissues. It is done under ultrasound guide.

Note: The recurrence rate of hydrocephalus is 2–5%.

SHOULDER DYSTOCIA

It means difficulty in delivery of the shoulders after delivery of the head. After delivery of the head it becomes retracted onto the perineum, the "turtle sign".

Incidence

It varies greatly and is about 1 in 300 vaginal deliveries. Macrosomia increases the incidence. In case of macrosomia, the incidence is 5% in absence of maternal diabetes, and 15% in the presence of maternal diabetes.

Aetiology (Risk Factors)

1) Large baby as in case of maternal obesity, maternal diabetes and postterm pregnancy; (2) unrotated shoulders; (3) pelvic deformity; (4) sometimes tumours of the thorax, abdomen and fetal ascites prevent descent of the body after delivery of the head; (5) mid forceps or ventouse delivery is associated with a higher incidence of shoulder dystocia; (6) previous history of shoulder dystocia.

Management

1. The patient is put in an exaggerated lithotomy position – McRoberts technique – and a large episiotomy is performed.

2. If the shoulders are unrotated, move the anterior shoulder towards the midline. This is followed by moderate traction on the head helped by suprapubic pressure by the assistant.
3. If this fails, bring down the posterior arm and apply moderate traction on the delivered arm and head.
4. Some may try the Woods screw method. Two fingers are inserted into the vagina and used to push the posterior shoulder anteriorly for 180° in a corkscrew fashion until it appears below the symphysis pubis. Now, traction is applied on the head to deliver the shoulders.
5. If the above measures fail, we fracture or cut the anterior clavicle (cleidotomy) to reduce the biacromial diameter, or we do symphysiotomy to deliver the shoulders, or the fetal head is grasped and pushed inside the vagina followed by immediate caesarean section (Zavanelli manoeuvre).

Complications

A) Fetal

1. Asphyxia and even death.
2. Injury of the brachial plexus of nerves and Erb's palsy as well as facial nerve injury.
3. Fracture of clavicle or humerus. Fracture of cervical spine is very rare, and is due to twisting of the fetal neck.

B) Maternal

1. Cervical, vaginal, and perineal lacerations, and extension of episiotomy.
2. Postpartum haemorrhage due to lacerations and uterine atony because of over-distension by the large fetus, and if manipulations take a long time.
3. Rupture of uterus may occur due to manipulations.

Note: In the exaggerated lithotomy position, the thighs are markedly flexed over the abdomen. This results in upward movement of the symphysis pubis and commonly releases the impacted anterior shoulder (McRoberts technique). The position also straightens the maternal lumbar lordosis and lumbosacral angle, thereby reducing the obstruction caused by the sacral promontory. In addition the angle of inclination of the pelvic inlet is reduced, making the plane of inlet perpendicular to the maternal expulsion forces.

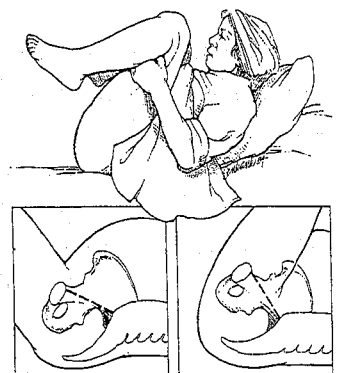


Fig. 99.2 McRoberts manoeuvre.

CONTRACTED PELVIS

Anatomical Definition

It is a pelvis in which one or more of its diameters is reduced by one centimeter or more below the normal.

Obstetrical Definition

It is a pelvis in which one or more of its diameters is reduced so that it interferes with the normal mechanism of labour.

However, the most important factor is the size of the fetal head in relation to the size of the pelvis. In fact, the head is the best pelvimeter.

Types of Female Pelvis

The non-diseased female pelvises include 4 main types (Caldwell and Moloy classification):

A) *The Gynaecoid Pelvis: (50%)*

It is the normal female pelvis. The inlet is circular or slightly transverse oval. The pelvic cavity is shallow and wide. The sacrosciatic notch is shallow and wide. The subpubic angle is wide 90–100°. The anterior surface of the sacrum is concave from above downwards and from side to side.

B) *The Android Pelvis: (20%)*

The inlet is triangular or heart shaped with a narrow anterior segment and a wide posterior segment. This favours occipito-posterior position. The promontory is projecting forwards. The pelvic cavity is deep and narrow. The sacrosciatic notch is deep and narrow. The subpubic angle is narrow 70° or less. The sacrum is flat, its lower end is inclined forwards. The ischial spines project inwards. This leads to funnelling of the pelvis.

C) *The Anthropoid pelvis: (25%)*

The inlet is a longitudinal oval, so the antero-posterior diameters of the inlet, cavity and outlet are longer than the transverse diameters. The sacrosciatic notch is wide. The subpubic angle is somewhat narrow. The sacrum is flat and usually has six segments.

D) *The Platypelloid or Simple Flat Pelvis: (5 %)*

The inlet is a transverse oval so the transverse diameters of the inlet, cavity and outlet are longer than the antero-posterior diameters. The subpubic angle is very wide.

Mixed types can occur and are more common than the pure forms.

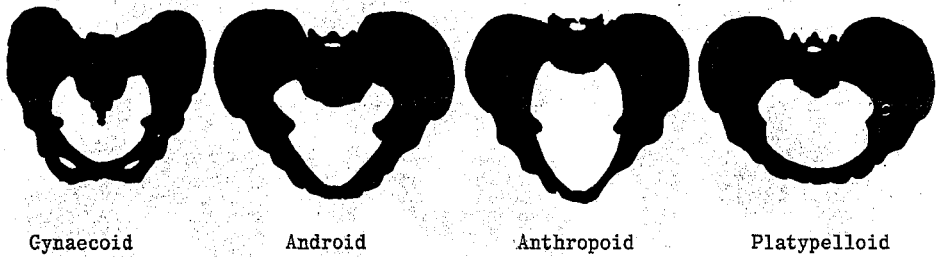


Fig. 100. Types of female pelvis

Aetiology of Contracted Pelvis

A) Congenital Contraction

1) Small gynaecoid pelvis (generally contracted pelvis); (2) small android pelvis; (3) small anthropoid pelvis; (4) small platypelloid pelvis; (5) Nägele pelvis (congenital absence of one ala of the sacrum); (6) Robert pelvis (congenital absence of both alae of sacrum); (7) high assimilation pelvis (sacrum made of six vertebrae); (8) low assimilation pelvis (sacrum made of four vertebrae); (9) deep acetabula (Otto pelvis).

B) Metabolic Diseases of Bone

1) Rickets; (2) osteomalacia (triradiate pelvis).

C) Diseases of the Spinal Column

1) Lumbar kyphosis; (2) scoliosis; (3) spondylolisthesis.

D) Diseases of Pelvic Bones

1) Osteoma; (2) fractured pelvis with callus formation.

E) Diseases of Lower Limbs

1) Shortening of one lower limb may lead to an obliquely contracted pelvis (asymmetrical pelvis); (2) poliomyelitis; (3) tuberculosis of hip joint.

Note: In lumbar kyphosis, the body weight pushes the base of the sacrum backwards so its tip moves forwards causing tunnel pelvis. In spondylolisthesis, the 5th lumbar vertebra and the vertebral column move to lie in front of the sacral promontory and this leads to rotation of the sacrum as in the kyphotic pelvis.

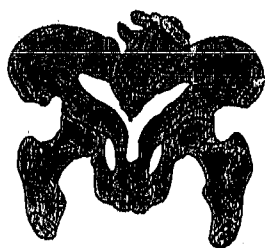


Fig. 101. Osteomalacic pelvis

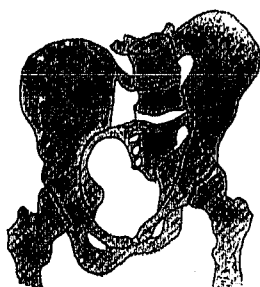


Fig. 102. Niegelo pelvis

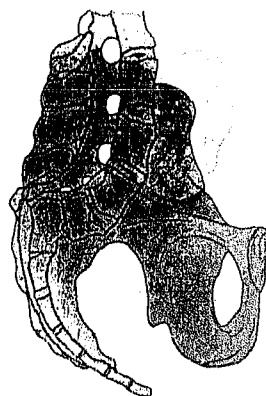


Fig. 103. Spondylolisthetic pelvis

Diagnosis of Contracted Pelvis (Cephalopelvic Disproportion)

Diagnosis includes history, general examination, abdominal examination, pelvimetry, estimation of fetal weight by ultrasound, and tests for cephalopelvic disproportion.

1. History

- a) History of rickets in childhood (delayed walking or delayed dentition).
- b) History of trauma or disease of the spine, pelvis or lower limbs.
- c) Bad obstetric history: There is a previous history of prolonged labour ending in still-birth or difficult forceps delivery or caesarean section for disproportion.

2. General Examination

- a) The general appearance; feminine suggesting a gynaecoid pelvis or masculine suggesting android pelvis. However, there is exception to this rule.
- b) Height: Short females below 150 cm depending upon the race, usually have contracted pelvis. Also, a shoe size 5 or less suggests pelvic contraction.
- c) Gait: Abnormal gait suggests abnormality in the pelvis or lower limbs.
- d) Signs of spinal deformity as kyphosis.
- e) Manifestations of old rickets as bow legs.
- f) The presence of dystocia dystrophia syndrome (page 212).

3. Abdominal Examination

a) Inspection

Pendulous abdomen in a primigravida is suggestive of contracted pelvis. Scar of previous caesarean section; suprapubic hair; feminine or masculine.

b) Palpation

Malpresentation or non engagement of the head in the last 2 weeks of pregnancy in a primigravida may be due to contracted pelvis.

4. Pelvimetry

The pelvic diameters can be evaluated by:

- a) Clinical pelvimetry.
- b) Radiological pelvimetry.
- c) Computed tomographic pelvimetry.
- d) Magnetic resonance imaging.

a) Clinical Pelvimetry

This includes inlet, outlet and internal pelvimetry.

Inlet Pelvimetry

Measurement of the interspinous, intercrystal and external conjugate diameters has no obstetric value as these are diameters of the false pelvis.

Outlet Pelvimetry

The intertuberos diameter is considered normal if it is over 8 cm. It can be roughly estimated by trying to place the closed fist against the perineum between the ischial tuberosities, after measuring the width of the closed fist. Usually the closed fist is wider than 8 cm. Sometimes the intertuberos diameter is narrow but vaginal delivery can still occur because of a long posterior sagittal diameter. So we have to apply Thom's rule. If the sum of the intertuberos diameter and posterior sagittal diameter is less than 15 cm, the outlet is contracted.

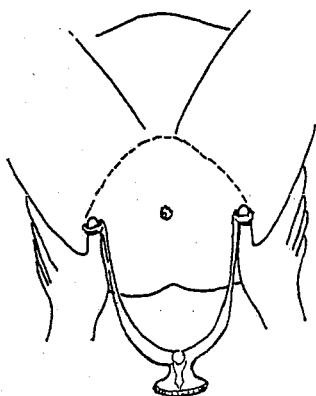


Fig. 104. Measurement of intertuberos diameter

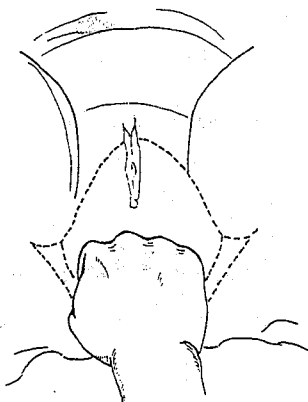


Fig. 105. Rough measurement of intertuberos diameter.

Internal Pelvimetry

Vaginal examination is performed at 36 weeks if the head is not engaged. Before that time the tissues are not soft enough to allow full exploration. The anterior surface of the sacrum is palpated from above downwards and from side to side. Note the distance between the ischial spines, and the size of the subpubic angle (normally 2 fingers can be placed immediately below the symphysis pubis). The diagonal conjugate is measured and subtract 1.5 cm to know the length of the true conjugate. Also the antero-posterior diameter of the outlet is estimated.

b) Radiological Pelvimetry

X-ray pelvimetry is no longer considered necessary in the management of cephalic presentation when pelvic contraction is suspected. It is needed in breech presentation and if there is a history of trauma or disease of the spine, pelvis, or lower limbs.

In pregnancy X-ray pelvimetry can be limited to a single lateral film. In the non-pregnant woman three views are taken; (a) view of the inlet; (b) view of the pelvic outlet, and (c) lateral view.

c) Pelvimetry Using Computed Tomography (CT Scanning)

Advantages: Less radiation exposure, more accurate and easier to perform. Cost comparable to X-ray pelvimetry.

d) Pelvimetry Using Magnetic Resonance Imaging (MRI)

Advantages: Absence of radiation, accurate measurements and complete imaging of the fetus. However, it is expensive and time consuming and available only in certain centers.

Note: The radiation dose of X-ray pelvimetry is about 1000 millirads (one rad) and 40–400 mrad from CT scanning.

5. Fetal Weight

Determined by ultrasound to diagnose macrosomia.

6. Tests for Cephalopelvic Disproportion

These are performed at 36 weeks if the head is not engaged, i.e. at the time of internal pelvimetry. These tests include the Pinard method or Muller-Kerr method.

Degrees of Cephalopelvic Disproportion

1. *Minor disproportion:* The anterior surface of the head is in line with the posterior border of the symphysis pubis. The head will engage during labour as a result of moulding and vaginal delivery is the rule.

2. *Moderate disproportion (first-degree disproportion):* The anterior surface of the head is in line with the anterior border of the symphysis pubis. Sufficient moulding of the head may occur during labour allowing vaginal delivery.
3. *Severe disproportion (second-degree disproportion):* The head projects beyond the anterior surface of the symphysis pubis (overriding or overhanging head). Vaginal delivery of a living baby is impossible.

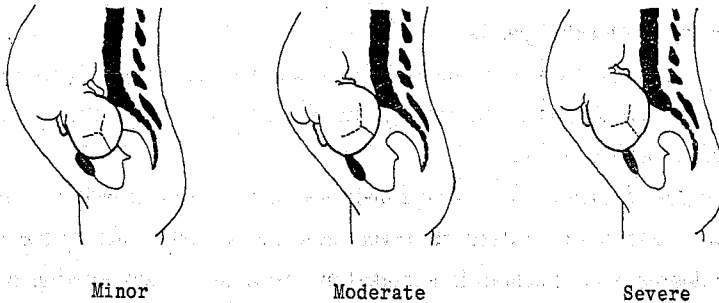


Fig. 106. Degrees of cephalopelvic disproportion

Anatomical Degrees of Contracted Pelvis

1. *Minor degree of contraction:* The true conjugate is between 9 and 10 cm. It corresponds with minor degree disproportion.
2. *Moderate degree of contraction:* The true conjugate is between 8 and 9 cm and corresponds with moderate degree disproportion.
3. *Severe degree of contraction:* The true conjugate is between 6 and 8 cm and corresponds with severe degree disproportion.
4. *Extreme degree of contraction:* The true conjugate is less than 6 cm, vaginal delivery of the fetus is impossible even after craniotomy, because the bimastoid diameter (7.5 cm) cannot be reduced even after crushing of the head.

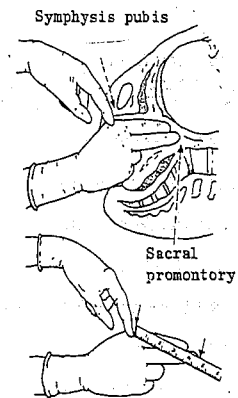


Fig. 107. Measuring the diagonal conjugate diameter

Notes:

1. The diagonal conjugate is measured by introducing the index and middle fingers of the right hand into the vagina. If the tip of the middle finger can reach the sacral promontory, a mark is made on the index finger at the lower border of the symphysis pubis. The distance is measured between the tip of the middle finger and the mark on the index one. The promontory cannot be reached in a normal pelvis.

2. Estimation of cephalopelvic disproportion:

- a) *Pinard method*: Bladder is evacuated. Patient in semisitting position (to bring the fetus perpendicular to the pelvic brim). The fetal head is grasped with the left hand and pushed downward and backward into the pelvis. The forefinger of the right hand palpates the upper border of the symphysis pubis to determine the degree of disproportion.
- b) *Muller-Kerr method*: Bladder is evacuated. Semisitting position with the hips and knees flexed. The head is grasped with the left hand and pushed downward and backward into the pelvis. The index and middle fingers of the right hand in the vagina will determine the degree of descent of the head and at the same time the thumb of the internal hand over the symphysis pubis determines the degree of disproportion.

Management of contracted pelvis

1. Minor degree of contraction or minor disproportion. Vaginal delivery is the rule.
2. Moderate degree of contraction or moderate disproportion. Treatment will be trial of labour, or caesarean section.
3. Severe degree of contraction or severe disproportion. Caesarean section if the fetus is alive and craniotomy if it is dead. However, caesarean section is safer for the mother.
4. Extreme degree of contraction. It is treated by caesarean section whether the fetus is alive or dead.
5. Treatment of contracted outlet according to Thom's rule.

Vaginal Delivery

This occurs in minor degree of contraction due to moulding of the head, relaxation of pelvic joints, stretching and thinning of the lower uterine segment and the bladder becoming an abdominal organ.

Caesarean Section

It is indicated in the following conditions:

1. Extreme degree of contraction whether the fetus is alive or dead.
2. Severe degree of contraction if the fetus is alive. Craniotomy is done if the fetus is dead. However, caesarean section is safer for the mother than craniotomy.
3. Moderate degree of contraction if trial of labour fails or is contraindicated.
4. Marked outlet contraction.
5. Elderly primigravida.
6. Nonvertex presentation: face, brow, breech or transverse lie.
7. Placenta praevia.

Trial of Labour

The patient is left for a reasonable time, one hour, in the second stage of labour (after full cervical dilatation) in the presence of good uterine contractions. If after such a trial the head fails to engage, caesarean section is performed. If the head engages, leave for spon-

taneous vaginal delivery. Trial of labour is carried out when the following conditions are fulfilled:

1. The patient should be a young healthy primigravida. If elderly primigravida caesarean section is done. If multigravida the previous labours were already a trial, unless the previous labours were preterm. The patient must be healthy because if there is heart disease or pre-eclampsia the stress of trial of labour is undesirable.
2. Moderate degree of contraction.
3. Vertex presentation.
4. Absence of placental insufficiency.
5. Absence of outlet contraction.

Technique of Trial of Labour

1. It is carried out in hospital because caesarean section may be needed.
2. As the head does not fit well against the lower uterine segment, trial of labour is frequently accompanied by premature rupture of membranes, prolapse of cord and uterine inertia. Guard against premature rupture of membranes by:
 - a) Keeping the patient in bed, no walking is allowed.
 - b) Avoid unnecessary vaginal examination.
3. Observation of the maternal pulse, temperature, blood pressure, fetal heart sounds, frequency and strength of uterine contractions.
4. When the membranes rupture a vaginal examination is performed to estimate the degree of cervical dilatation, to exclude prolapse of cord and to know the position of the head. Trial of labour starts when the cervix is fully dilated.

Management of Contracted outlet

According to Thom's rule: If the sum of the intertuberos diameter and the posterior sagittal diameter is less than 15 cm, caesarean section is done if the fetus is alive and craniotomy if dead. If the sum of the two diameters is 15 cm or more allow for vaginal delivery. If the head becomes arrested apply the forceps and perform a deep episiotomy with the patient in exaggerated lithotomy position (McRoberts position). If the forceps fails and the fetus is alive do caesarean section and if dead do craniotomy. Symphysiotomy can be done alternative to caesarean section.

Complications of Contracted Pelvis

I. Maternal

A) During Pregnancy

1. If the uterus is retroverted and the sacral promontory is projecting forwards, this may lead to abortion, incarceration or anterior sacculation of the uterus.

2. Malpresentation.
3. Nonengagement of the presenting part.
4. Pendulous abdomen due to nonengagement of the presenting part.
5. Pyelonephritis of pregnancy due to a high assimilation pelvis causing pressure on the ureters.

B) During Labour

1. Liability to premature rupture of membranes (the presenting part is not fitting well against the lower uterine segment so the increased intra-amniotic pressure resulting from uterine contraction is entirely transmitted to the bag of forewaters).
2. Liability to prolapse of cord, arm or both as in the flat pelvis.
3. Uterine inertia, slow dilatation of the cervix and prolonged labour. The presenting part is not fitting well against the lower uterine segment leading to absence of reflex polarity.
4. Obstructed labour and rupture of uterus.
5. Necrotic fistulae due to prolonged compression of the soft tissues between the fetal head and pelvic walls (vesicovaginal, vesicocervical and rectovaginal).
6. Postpartum haemorrhage due to inertia and lacerations.
7. Puerperal sepsis due to prolonged labour, premature rupture of membranes and increased incidence of interference.
8. Injury of pelvic joints or nerves due to difficult forceps delivery. Fracture of the coccyx.

II. *Fetal*

1. Asphyxia due to prolonged labour, obstructed labour and compression of prolapsed cord.
2. Formation of a large caput succedaneum.
3. Fracture of the skull bones.
4. Intracranial haemorrhage due to excessive moulding. Slight moulding reduces the biparietal diameter by about 0.5 cm and is not dangerous.
5. Congenital fetal pneumonia and fetal septicaemia due to prolonged labour or premature rupture of membranes.

Note: Reflex polarity means pressure on the lower uterine segment and cervix by the presenting part, reflexly stimulates the uterus to contract.

THE FLAT PELVIS

It is a pelvis in which the antero-posterior diameter of the inlet is shorter than the transverse diameter. There are two types of flat pelvis: (a) the simple flat pelvis, and (b) the rachitic flat pelvis.

Mechanism of Labour

1. The head descends with the sagittal suture in the transverse diameter of the inlet.
2. The head engages by asynclitism to enter the pelvis by a smaller diameter which is the supraparietal-subparietal diameter (9 cm).
3. Lateral displacement of the head occurs to bring the small bitemporal diameter (8 cm) into the short antero-posterior diameter and the biparietal diameter into the wide lateral part of the pelvis.
4. Deflexion: Even by the above mechanism the biparietal diameter will not descend leading to deflexion of the head. Exaggeration of deflexion may lead to brow or face presentation.
5. Further descent of the head occurs by correction of asynclitism.
6. In the rachitic flat pelvis once the head has passed below the pelvic inlet it will be delivered by the normal mechanism because the outlet is not contracted. In the simple flat pelvis the antero-posterior diameter of the outlet is also contracted and this interferes with internal rotation of the head leading to deep transverse arrest in the majority of cases.
7. Premature rupture of membranes and prolapse of cord are common.

FUNNEL PELVIS (CONTRACTED PELVIC OUTLET)

It is a pelvis in which the capacity diminishes from the inlet to the outlet so the main feature is contraction of the outlet. Contracted outlet is present in the following conditions:

- 1) Android pelvis; (2) anthropoid pelvis; (3) lumbar kyphosis; (4) spondylolisthesis; (5) high assimilation pelvis; (6) oblique pelvis; and (7) osteomalacia.

Management of Contracted Outlet: see before.

OBLIQUE (ASYMMETRICAL) PELVIS

One side of the pelvis is normal while the other side is contracted, so one oblique diameter is shorter than the other. The causes are: (1) Nägele pelvis, (2) scoliosis, (3) fracture or tumour affecting one side of the pelvis, (4) disease affecting one lower limb as shortening, poliomyelitis, and tuberculosis of hip joint.

TRAUMATIC OBSTETRIC LESIONS

I. PERINEAL LACERATIONS

Aetiology

1. Obstetric trauma which is the commonest cause.
2. Other types of trauma as falling on sharp objects and defloration injuries.

Causes During Labour

A) Overstretching of the Perineum due to

1. Bad management of the second stage of labour by allowing extension of the head before crowning.
2. Delivery of the head as face to pubis.
3. Delivery of the head as mento-anterior.
4. Large head or shoulders.
5. Narrow subpubic arch pushing the head posteriorly.
6. Narrow vagina.
7. Forceps delivery.

B) Rapid Stretching of the Perineum

1. Precipitate labour.
2. Rapid delivery of the aftercoming head in breech.

C) Rigidity of the Perineum

1. Elderly primigravidae.
2. Perineal scar due to previous operation.
3. Oedema of the vulva as in pre-eclampsia so the tissues are friable.

Degrees of Perineal Tears

First Degree

The perineal skin and the mucosa of the posterior vaginal wall are torn.

Second Degree

The tear involves the above structures as well as the muscles of the perineal body but does not involve the external anal sphincter.

Third Degree

The tear involves the external anal sphincter.

Fourth Degree

The rectal wall is torn.

First and second degrees are called incomplete tears, while third and fourth degrees are complete tears.

Hidden Perineal Tear

The levator ani muscles become lacerated as a result of overstretching of the perineum, without a visible tear in the vaginal mucosa or perineal skin. Such cases may be followed by prolapse.

Complications of Perineal Tears

1. Haemorrhage (postpartum haemorrhage).
2. Infection.
3. Prolapse.
4. Stress incontinence.
5. The vagina becomes dilated and this predisposes to vaginitis and leucorrhoea.
6. In case of complete tear there is loss of voluntary control over the passage of faeces and flatus.
7. Incomplete healing of a perineal tear extending to the rectum leads to a rectovaginal fistula.
8. Dyspareunia from a tender scar in the vagina.

Treatment

A) Prophylactic Treatment

Proper management of the second (perineal) stage of labour.

B) Surgical Treatment

Any perineal tear whatever small, should be repaired immediately or maximally within 24 hours. Later than this it is considered as a septic wound and repair is done after 3-6 months when infection has disappeared and the tissues become involuted.

1. Incomplete Tear (First and Second Degrees)

Repair is usually done under local infiltration anaesthesia as 1% lignocaine solution.

2. Complete Tear (Third and Fourth Degrees)

Repair is done in the operating theatre under general anaesthesia.

a) The wall of the rectum and anal canal is closed from above downwards by thin chromic catgut or delayed-absorbable suture as Vicryl or Dexon. Interrupted inverting Lambert sutures are used to close the mucosa; (b) the torn ends of the external anal sphincter are brought together anterior to the anus by two stitches; (c) the levator ani muscles are approximated by interrupted sutures; (d) the vagina is closed by interrupted sutures; (e) the superficial perineal muscles are then approximated in the midline; (f) the perineal skin is closed by interrupted sutures or subcuticular Vicryl or Dexon stitches.

Postoperative Care of A Complete Perineal Tear

1. The perineal wound is kept clean and dry. After each micturition or defecation, the perineum is washed with antiseptic solution as Savlon, and covered by a sterile vulval pad.
2. Nothing by mouth for 48 hours, only intravenous fluids are given. Then oral fluids are given for 3 days. Milk is not given to avoid distension.
4. Antibiotics to guard against wound infection.
5. On the fifth night give a castor oil purge (60 ml). Then the patient is given liquid paraffin daily for 2 weeks to avoid constipation.
6. If silk sutures were used they are removed on the 6th day.
7. No sexual intercourse for 3 months.
8. In subsequent delivery a prophylactic episiotomy must be done.

II. CERVICAL LACERATIONS

Aetiology

These are the result of labour, few cases occur after surgical dilatation of the cervix as in the treatment of spasmodic dysmenorrhoea. The causes of lacerations during labour are: (1) forceps extraction or breech extraction before the cervix is fully dilated; (2) precipitate labour; (3) misuse of oxytocin; (4) large baby; (5) annular detachment of the portio vaginalis may occur when the external Os fails to dilate (cervical dystocia); (6) fibrosis of the cervix makes it more liable to tear.

Types

Laceration may be unilateral, bilateral or multiple extending radially from the external Os (stellate laceration). Laceration is usually on the left side, because the occiput is commonly on the left side and because of right obliquity of uterus. Bilateral vertical laceration causes eversion of the cervical lips and ectropion.

Diagnosis

1. The presence of postpartum haemorrhage while the uterus is well contracted makes one suspect a laceration in the genital tract.
2. Bimanual examination may detect the presence of the tear.
3. The cervix is exposed by inserting a self-retaining posterior vaginal speculum (Auvard speculum) and the anterior and posterior lip of the cervix is grasped by a ring forceps to allow inspection of the whole cervix. Vaginal retractors help to give complete exposure.

Complications

1. The laceration may involve the cervical branch of the uterine artery leading to severe haemorrhage.
2. Infection may occur leading to acute or chronic cervicitis or infection may spread causing parametritis.
3. A deep laceration extending to the internal Os is liable to cause habitual abortion or preterm labour (incompetent cervix).
4. The laceration may extend upward to involve the lower uterine segment leading to uterine rupture.
5. Injury of the ureter from extension of a lateral tear or during its repair.

Treatment

When a cervical laceration occurs it is sutured immediately. If it is associated with infection, the infection is treated at first followed by trachelorrhaphy. Incompetent cervix causing habitual abortion or preterm labour is treated by Shirodkar or McDonald operation done during pregnancy.

III. RUPTURE OF THE UTERUS**Incidence**

It varies between 1:1000 to 1:3000 deliveries according to the standard of obstetric practice. 95% of cases occur in multiparae and this is due to: (1) degeneration and weakness of uterine muscle caused by repeated deliveries; (2) increased incidence of malpresentation due to laxity of uterine and abdominal muscles; and this causes obstructed labour; (3) pendulous abdomen is common due to laxity of uterine and abdominal muscles leading to misdirection of the uterine force so the fetus is pushed against the posterior wall of uterus; (4) the size of the fetus tends to increase with parity; (5) osteomalacia may affect the pelvis with repeated pregnancies; leading to obstructed labour; (6) the obstetrician may have a false sense of security from the history of previous normal deliveries.

Aetiology

Rupture may occur during pregnancy or labour.

I. During Pregnancy

A) Spontaneous Rupture

1. Rupture of a scar in the uterus due to previous caesarean section, myomectomy or previous perforation.
2. Abruptio placentae (concealed type).
3. Sacculation; anterior wall sacculation in case of incarcerated retroverted gravid uterus, or posterior sacculation after ventrofixation of the uterus.
4. Rupture of angular pregnancy or pregnancy in a rudimentary horn.
5. Invasive mole (chorioadenoma destruens).

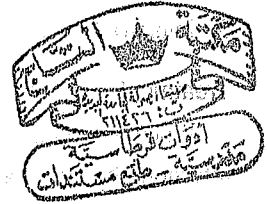
B) Traumatic Rupture

1. Kick or blow on the abdomen.
2. If excessive force is used during external cephalic version.

II. During Labour

A) Spontaneous Rupture

1. Rupture of a scar in the uterus.
2. Abruptio placentae.
3. Multiparity; due to the causes mentioned before.
4. Sacculation of uterus.
5. Obstructed labour from any cause, e.g. contracted pelvis.
6. Maluse of oxytocin drip.
7. An old cervical tear may extend upwards with the onset of uterine contractions.



B) Traumatic Rupture

Examples include:

1. Forceps or breech extraction before the cervix is fully dilated.
2. Internal podalic version after drainage of the amniotic fluid.
3. Destructive operations as craniotomy or decapitation.
4. During manual removal of the placenta.

Note: The commonest cause of uterine rupture is rupture of a previous caesarean section scar.

Pathology

I. Type of Rupture

- a) *Complete rupture*: The tear involves all the layers of the uterus including the peritoneum. The uterine cavity becomes continuous with the peritoneal cavity. Rupture of the upper uterine segment is usually complete because the peritoneum is adherent to the muscle layer.
- b) *Incomplete rupture*: The tear does not involve the peritoneal coat which remains intact. Rupture of the lower uterine segment is usually incomplete.

II. Site of Rupture

This depends upon the cause of rupture.

- a) In case of obstructed labour, the tear is in the lower uterine segment. It is transverse or oblique. It is more often on the left side due to right obliquity of the uterus and the occiput is commonly to the left side.
- b) In case of ruptured scar the tear occurs at the site of the scar.

III. The Bleeding Associated with Rupture

It is variable, if the uterine artery or one of its main branches is torn severe bleeding occurs. If the fetus and placenta escape into the peritoneal cavity, the empty uterus retracts and bleeding may be slight.

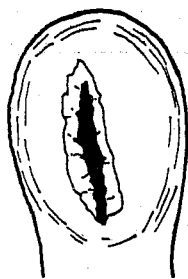


Fig. 108. Rupture of a classical
Caesarean scar



Fig. 109. Transverse rupture
of lower segment

Note: *Uterine dehiscence* means separation of the edges of a uterine scar, however, the peritoneum overlying the scar and the fetal membranes remain intact and the fetus is not extruded into the peritoneal cavity. Bleeding is absent or minimal.

Clinical Picture

1. Rupture During Pregnancy

There is usually a history of trauma to the abdomen or previous operation on the uterus. There is a sudden onset of severe abdominal pain, pain may be felt in the shoulder on lying down (blood under the diaphragm irritates the phrenic nerves) and vaginal bleeding may occur.

General Examination

There is a variable degree of shock depending upon the amount of blood loss and the extent of the tear.

Abdominal Examination

If the fetus escapes into the peritoneal cavity, the fetal parts will be easily felt and the fetal heart sounds will not be heard. The fetus is usually in an abnormal position as high transverse lie. The retracted uterus is felt as a firm pelvi-abdominal mass to one side of the fetus. In case of severe internal haemorrhage, shifting dullness may be detected, and rarely there is a bluish coloration around the umbilicus (Cullen sign).

Vaginal Examination

It may reveal vaginal bleeding.

Silent Rupture

In some cases the symptoms are mild and the patient comes walking to the hospital. This is liable to occur with rupture of a uterine scar as the bleeding is slight due to fibrosis (uterine dehiscence).

2. Rupture due to Obstructed Labour

The patient shows the picture of obstructed labour. Suddenly, she feels severe abdominal pain and feels something giving way. This is followed by cessation of uterine contractions. Pain may be felt in the shoulder on lying down and vaginal bleeding may occur.

General Examination: as before.

Abdominal Examination: as before.

Vaginal Examination

There is evidence of obstructed labour as oedema of the vulva, warm and dry vagina. There is recession of the presenting part or it is freely mobile. Vaginal bleeding may be present and the tear may be felt.

N.B.: If rupture is incomplete the symptoms may be slight and uterine contractions may continue after the rupture.

3. Traumatic Rupture During Labour

- a) The condition is suspected if shock or haemorrhage occurs after delivery.
- b) If the placenta escapes into the peritoneal cavity, it will be retained, and the rupture is discovered during manual removal of the placenta.
- c) The condition may pass unnoticed until peritonitis and ileus develop.

Differential Diagnosis

All causes of acute abdomen including abruptio placentae.

Treatment

I. Prophylactic

A) During Pregnancy

1. Antenatal care to detect any fetal or maternal abnormality which may lead to obstructed labour.
2. The grand multipara should deliver in hospital.
3. Patient with upper segment caesarean section scar must be delivered by elective section at 38 weeks.

B) During Labour

1. Proper use of oxytocin.
2. Version is contraindicated if the amniotic fluid is drained.
3. No forceps or breech extraction before full dilatation of the cervix.
4. Exploration of uterine cavity is done after delivery in the presence of a uterine scar and after instrumental or difficult delivery.

II. Active Treatment

Anti-shock measures are given followed by immediate laparotomy. The fetus and placenta are removed and hysterectomy is performed (supravaginal hysterectomy). However, if there is a ruptured scar with clean cut edges, or if the patient is young or childless, the tear is sutured. In this case, pregnancy is avoided for one year and when

pregnancy occurs the patient is hospitalized 4 weeks before term and caesarean section is done 2 weeks before the expected date.

Note: Repair plus tubal sterilization may be done to conserve the psychological effect of menstruation.

Complications

I. Maternal

The maternal mortality is 0–10%, due to shock, haemorrhage, infection (peritonitis and parametritis) and paralytic ileus. Shock is the commonest cause of death. Amniotic fluid embolism may occur if amniotic fluid is forced into the torn blood vessels. Injury of the bladder, ureter or other abdominal organs may complicate rupture of uterus, and we must exclude these injuries during operation.

II. Fetal

Fetal mortality is 50–75%, due to placental separation leading to fetal asphyxia.

ANTEPARTUM HAEMORRHAGE (APH)

Definition

It is bleeding from the genital tract occurring after 20 weeks of pregnancy and during labour before delivery of the fetus (first and second stages of labour are included).

Aetiology

1. Bleeding from the Placental Site

- a) Abruptio placentae (accidental haemorrhage). Bleeding due to premature separation of a normally situated placenta.
- b) Placenta praevia. Bleeding due to premature separation of an abnormally situated placenta.

2. Bleeding from Extraplacental Site (Incidental Haemorrhage)

In few cases (about 5%) bleeding is due to a local lesion in the genital tract as cervical erosion, carcinoma of cervix and rupture of uterus. Vasa praevia and bloody show are other causes.

Note: Abruptio placentae is the commonest cause of APH.

VASA PRAEVIA

It is a rare condition in which the umbilical vessels run through the membranes over the region of the internal Os of the cervix. It occurs with velamentous insertion of the cord, with placenta succenturiata, and with bipartite placenta. It is the only cause of APH of fetal origin.

Incidence

About 1 in 5000 deliveries.

Diagnosis

A) Before rupture of membranes

1. The vessels may be palpated in the membranes during vaginal examination.
2. Sonography. If it diagnoses placenta with accessory lobe or bipartite placenta, we suspect the presence of vasa praevia. Transvaginal colour Doppler ultrasound may show the aberrant vessels.

B) After rupture of membranes

There is vaginal bleeding associated with fetal bradycardia.

The blood can be tested for fetal haemoglobin by the Apt test. However, the test is time-consuming and not practical.

Treatment

The fetus is delivered immediately. If the cervix is fully dilated apply the forceps or ventouse. Caesarean section is performed when the cervix is not fully dilated and when conditions are not suitable for vaginal delivery. The fetal mortality is 50-75%.

Notes for the postgraduate:

- Haemorrhage from vasa praevia can occur without rupture of membranes in about 25% of cases. Leakage occurs from the blood vessels as they are not surrounded by Wharton jelly.
- Apt test. One part of vaginal blood is mixed with 5 parts of tap water and the mixture is centrifuged for 2 minutes. The supernatant is then mixed with an equal part of 1% sodium hydroxide solution. Centrifuge again for 2 minutes and observe the colour. If the solution remains pink it indicates fetal blood as fetal haemoglobin is not denatured by alkali. If the solution becomes yellow or brown it is maternal blood as the maternal haemoglobin is denatured by alkali to form alkaline haematin.
- Fetal haemoglobin is tested for by Apt test, by Kleihauer test to detect fetal red cells in maternal blood in case of erythroblastosis fetalis, and by haemoglobin electrophoresis.

PLACENTA PRAEVIA (P.P.)

Definition

The placenta is partially or completely inserted in the lower uterine segment.

Incidence

About 0.5% of pregnancies.

Parity

More common in multigravidae than primigravidae (4:1).

Predisposing Factors

The common *predisposing factors* are increased maternal age (above 35), increased parity, multiple pregnancy, previous caesarean section, previous P.P., and smoking. The recurrence rate after one P.P. is about 5%.

Aetiology

1. *Basal Theory*: The fertilized ovum becomes embedded in the lower uterine segment due to (a) delayed disappearance of the corona radiata and zona pellucida; or (b) delayed development of the chorionic villi or (c) chronic endometritis with a poor decidual reaction so the placenta spreads over a wide area and so it is more in multigravidae.
2. *Capsular Theory* (Hoffmeir): The condition is due to growth of the chorionic villi in the decidua capsularis and its development into placenta.

3. *Abnormally large placenta* as in case of twins and polyhydramnios.

Grades (Types) of Placenta Praevia

1. *Grade 1* (type 1, low-lying placenta or P.P. lateralis). The lower edge of the placenta reaches the upper part of the lower uterine segment. The term lateralis is not correct because the placenta may be anterior or posterior.
2. *Grade 2* (type 2 or P.P. marginalis). The lower edge of the placenta reaches down to the margin of the internal Os of the cervix but does not cover it.
3. *Grade 3* (type 3, or incomplete P.P. centralis). The placenta covers the internal Os when it is closed, but covers it partially when it is fully dilated.
4. *Grade 4* (type 4, or complete P.P. centralis). The placenta covers completely the internal Os when it is fully dilated.

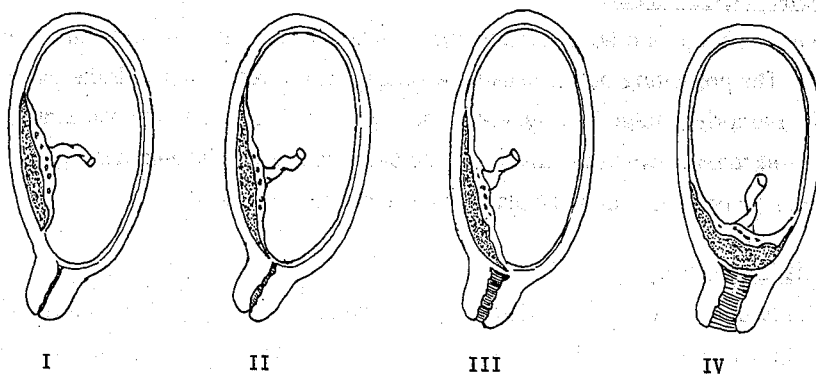


Fig. 110. Types of placenta praevia

The Mechanism of Bleeding

Stretching of the lower uterine segment normally occurs during pregnancy and labour. The placenta is not elastic and cannot stretch and this leads to separation of the placenta from the lower uterine segment and bleeding, so bleeding is inevitable or unavoidable. In case of placenta praevia centralis bleeding may occur early in pregnancy and is usually severe because a great part of the placenta is attached to the lower uterine segment.

Diagnosis

I. Symptoms

There is vaginal bleeding after 20 weeks of pregnancy which is causeless, painless and recurrent. It is causeless but it may occur after coitus or vaginal examination. It is painless

unless it occurs during labour. It is usually recurrent because the lower uterine segment undergoes intermittent stretching during pregnancy causing intermittent separation of the placenta. The bleeding may not recur in type 1 P.P. (because the part attached to the lower segment may separate once leading to a single attack of bleeding). In fact any painless bleeding in the second half of pregnancy is assumed to be due to P.P. until proved otherwise.

II. Signs

A) General Examination

The general condition of the patient is related to the amount of blood loss because all the bleeding is revealed.

B) Abdominal Examination

The uterus is soft and not tender. Malpresentations as breech or shoulder are common (25–30%). The presenting part is usually not engaged and freely mobile. If the placenta is situated anteriorly a bulge may appear in the suprapubic region and the placenta may be felt as a soft mass between the hand and the presenting part. The fetal heart sounds are heard unless more than half of the placenta has separated.

C) Vaginal Examination

It is only done when it is decided to deliver the patient and placental localization by ultrasound was not done. The aim is to diagnose placenta praevia and this is followed immediately by caesarean section or rupture of the membranes (amniotomy) according to the findings. Examination must be done in the operating theatre under general anaesthesia with every thing ready for caesarean section and blood available for transfusion as severe bleeding may occur. Two teams of obstetricians are available; one performs vaginal examination and the other is scrubbed and gowned to proceed with immediate caesarean section if necessary (a double setup examination).

Note: Findings on vaginal examination:

1. If the cervix is closed a soft pulsating mass may be felt between the fingers in the vagina and the presenting part.
2. If the cervix is opened, the index finger is introduced and rotated to palpate the lower uterine segment all around to feel the placenta which is felt as a fibrous mass. A blood clot is soft and friable and breaks down easily under the examining finger.

III. Localization of the Placenta

- a) *Ultrasonography.* It is a safe and accurate method. It diagnoses P.P. in 95–98% of cases. If a low-lying placenta is diagnosed in early pregnancy, ultrasound is repeated every month because in the majority of cases (90%) the placenta migrates away from the

internal Os (migrating placenta). This migration is due to stretching of the lower uterine segment in late pregnancy. Migration will not appear after 32 weeks of pregnancy.

- b) *Soft tissue placentography*. Can be used if ultrasound is not available, a lateral film of the lower abdomen is taken with soft (low-voltage) X-rays.
- c) *Magnetic resonance imaging (MRI)*. It is the most precise method for placental localization. However, the method is expensive and only available in few centers

Differential Diagnosis

Other causes of antepartum haemorrhage: (1) abruptio placentae; (2) vasa praevia; (3) local lesions in the genital tract as cervical carcinoma. These are excluded by speculum examination.

Treatment

1. Treatment at Home

- a) A detailed history is taken. The patient is examined generally and abdominally, but no vaginal examination and no vaginal pack.
- b) A sedative as pethidine is given I.M. if there is pain or irritability. In case of severe bleeding intravenous fluids and other antishock measures are given.
- c) A sterile vulval pad is applied and the patient is transferred to hospital.

2. Treatment at Hospital

The case is fully investigated to diagnose the cause of bleeding including ultrasound to locate the placenta. Complete blood count, and blood group (ABO and Rhesus) are determined. Crossmatched blood is kept available. The treatment is according to the following plan:

i) The patient is not in labour

- a) If the bleeding is severe caesarean section is done.
- b) If the bleeding is slight or moderate and duration of pregnancy is less than 38 weeks conservative treatment is indicated. If pregnancy has reached 38 weeks we terminate pregnancy by caesarean section or amniotomy.

ii) The patient is in labour

- a) If the bleeding is severe caesarean section is done.
- b) If the bleeding is slight or moderate the treatment is amniotomy or caesarean section. However, if the placenta was not localized by ultrasound, vaginal examination is

performed in the operating theatre with all the precautions mentioned before followed by caesarean section or amniotomy.

Criteria for Amniotomy

The following conditions must be fulfilled:

1. The bleeding is slight or moderate.
2. Type I placenta praevia.
3. Vertex presentation.
4. The cervix is partially dilated to allow rupture of membranes.

If the method fails to stop the bleeding caesarean section is done. Amniotomy stops the bleeding by allowing descent of the head and compression of the placenta. Also rupture of the membranes will prevent further separation of the placenta from the uterine wall during uterine contraction. Amniotomy is done by the Amnihook or by a toothed instrument as the volsellum or Kocher.

Indications of Caesarean Section

1. Type 2, 3 and 4 placenta praevia.
2. Severe haemorrhage whether the patient is in or not in labour.
3. Elderly primigravida.
4. Any degree of contracted pelvis.
5. Nonvertex presentation.
6. If bleeding continues after amniotomy.

It is a lower segment caesarean section to allow better control of bleeding from the placental site and to leave a strong scar that can allow subsequent vaginal delivery.

Conservative Treatment

It is indicated if the patient is not in labour, bleeding slight or moderate, and pregnancy is less than 38 weeks. The aim is to prolong pregnancy until the fetus is mature enough to survive after delivery.

1. The patient is kept in hospital till delivery.
2. Treatment of anaemia.
3. Sonography to locate the site of the placenta, to determine the gestational age, presentation and the presence of congenital anomalies.
4. Speculum examination if sonography did not show placenta praevia to exclude local lesions.
5. The mother is given anti-D immunoglobulin after the first attack of bleeding if she is Rh negative. Injection is repeated every 6 weeks if intermittent bleeding continues.

6. Dexamethasone is given to stimulate fetal lung maturity if duration of pregnancy is less than 34 weeks. It can be repeated weekly. However, the value of repeating the dose is not certain.
7. Because of the possibility of placental insufficiency, the fetal growth and well-being are monitored (serial ultrasound and nonstress test).
8. Pregnancy is terminated at 38 weeks by caesarean section or amniotomy.

Note: The dangerous P.P. is an old term which was used in the past to describe P.P. marginalis situated posteriorly. In this case a large area of the placenta will be compressed between the head and the sacrum leading to fetal asphyxia if vaginal delivery is allowed. Now the treatment of P.P. marginalis is caesarean section whether situated anterior or posterior.

Complications of Placenta Praevia

I. Maternal Complications

A) During Pregnancy

1. Abortion.
2. Preterm labour due to premature separation of the placenta.
3. Premature rupture of membranes.
4. Antepartum haemorrhage.
5. Malpresentation.
6. Nonengagement of the presenting part.

B) During Labour

1. Premature rupture of membranes may occur due to malpresentation.
2. Liability to prolapse of cord (low implantation of the placenta).
3. Obstructed labour due to malpresentation.
4. Intrapartum haemorrhage.
5. Uterine atony secondary to anaemia.
6. Postpartum haemorrhage. The causes are: (a) weak contraction and retraction in the lower uterine segment; (b) uterine atony due to anaemia resulting from haemorrhage; (c) tears of the cervix and lower uterine segment easily occur because of increased vascularity and softening; (d) placenta accreta is frequent leading to retained placental fragments; (e) large placental site from which bleeding can occur.
7. Placenta accreta. The chorionic villi penetrate deeply into the uterine wall due to a poor decidual reaction in the lower uterine segment.
8. Retained placenta (due to atony and placenta accreta).
9. Lacerations of the cervix and soft lower uterine segment easily occur.
10. Air embolism may occur as the placental site is near the vagina.

C) During the Puerperium

1. Puerperal sepsis due to large placental area, the placental site is near the vaginal flora, occurrence of lacerations in the cervix and soft lower segment and diminished resistance to infection because of blood loss and anaemia.
2. Subinvolution of uterus.
3. Secondary anaemia.

Causes of Maternal Death (less than 1%)

1. Haemorrhage which may be ante-, intra- or postpartum. Usually the latter which kills the patient.
2. Air embolism.
3. Puerperal sepsis.

II. Fetal Complications

1. Prematurity and its complications as respiratory distress syndrome.
2. Increased incidence of malformation (4%) due to poor decidual reaction in the lower uterine segment.
3. Intrauterine growth restriction due to placental insufficiency.
4. Asphyxia and death due to premature separation of the placenta, compression of prolapsed cord and maternal hypoxia due to haemorrhage.

The above complications can lead to fetal death and prematurity is the main cause. The perinatal mortality is 5% or higher.

ABRUPTIO PLACENTAE (ACCIDENTAL HAEMORRHAGE)

Definition

It is antepartum haemorrhage due to premature separation of a normally situated placenta.

Incidence

About 1% of pregnancies.

Parity

The incidence increases with parity and maternal age.

Aetiology

1. Idiopathic: In most cases the cause is unknown.
2. Maternal hypertension due to any cause (30%).
3. Trauma: As a blow on the abdomen or during external cephalic version (1–5%).

4. Traction on the placenta by a short cord (absolute or relative).
5. Thrombosis of uterine or ovarian veins.
6. Torsion of the pregnant uterus.
7. Tension and anxiety states.
8. Deficiency of folic acid. This is not definitely proven.
9. Sudden drop in the intrauterine pressure after rupture of membranes in polyhydramnios or delivery of the first fetus in twins.
10. Occlusion of inferior vena cava as in the supine hypotensive syndrome. This leads to rise in the venous pressure in the retroplacental veins and their rupture.
11. Premature rupture of membranes predisposes to abruptio placentae, and vice versa.
12. Smoking (by causing vasospasm, ischaemia and decidual necrosis).
13. Cocaine use (leading to vasoconstriction and transient hypertension).
14. Regular aspirin intake.
15. The presence of antiphospholipid antibodies (lupus anticoagulant and anticardiolipin antibodies).
16. Circumvallate placenta.
17. The presence of uterine fibroid beneath the placenta.
18. Previous history of placental abruption. The risk of recurrence is about 10%.

Types

1. *Revealed haemorrhage*: The blood appears externally as vaginal bleeding.
2. *Concealed haemorrhage*: All the blood is retained inside the uterus. It is retained because of (a) atony of the uterine muscle due to uterine pathology in severe cases of pre-eclampsia, and it is the main cause; (b) abnormal adhesion of the placental margin; (c) abnormal adhesion of the membranes at the internal Os; (d) close fitting of head against lower uterine segment; (e) passage of blood inside the amniotic sac.
3. *Combined or mixed type*: The blood is partially concealed and partially revealed. It usually starts as concealed haemorrhage but when uterine contractions occur some of the blood appears externally as vaginal bleeding.

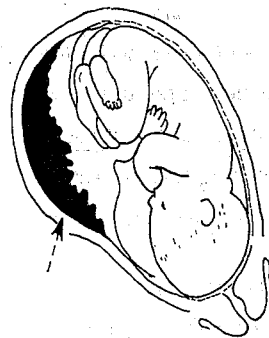


Fig. 111. Concealed haemorrhage

Pathology of Abruption Placentae

Bleeding leads to partial or complete separation of the placenta. The blood collecting behind the placenta leads to more placental separation and more bleeding. Occasionally the blood escapes between the muscle fibres of the myometrium giving the uterus a purple colour. This condition is known as Couvelaire uterus or uteroplacental apoplexy. The blood may even reach beneath the peritoneal coat producing areas of ecchymoses and the peritoneum may become fissured leading to intraperitoneal haemorrhage. Disseminated intravascular coagulation may complicate severe cases of placental abruption associated with fetal death (about 30%). In pre-eclampsia haemorrhage is liable to occur if systolic blood pressure reaches 160 mmHg or above.

Diagnosis

I. Concealed Haemorrhage

Symptoms

There is a sudden onset of severe abdominal pain. There may be a history of trauma or pre-eclampsia.

Signs

General Examination

- a) Signs of shock and internal haemorrhage as rapid pulse and subnormal temperature. The blood pressure is low but it may be normal due to a fall from a previous high level. Shock is haemorrhagic due to blood loss and neurogenic due to myometrial damage, stretching and fissuring of the peritoneal coat of the uterus.
- b) Signs of pre-eclampsia in the form of oedema and proteinuria are usually present.

Abdominal Examination

- a) The uterus is larger than the period of amenorrhoea and it gradually increases in size.
- b) The uterus is very tender and hard in consistency (board like).
- c) The fetal parts are not felt due to abdominal rigidity.
- d) The fetal heart sounds are absent and if present they show marked fetal distress (bradycardia and prolonged decelerations).

Vaginal Examination

There is no vaginal bleeding.

Investigations

1. Urinalysis for proteinuria.

2. Sonography. It can show a retroplacental or subchorionic haematoma.
3. Coagulation profile. Tests are done to diagnose disseminated intravascular coagulation (DIC) and coagulation failure. Tests are repeated every 2 hours.

II. Revealed Haemorrhage

Symptoms

The patient complains of vaginal bleeding. There may be a history of trauma or pre-eclampsia.

Signs

General Examination

- a) The general condition of the patient is related to the amount of blood loss because all the blood is revealed.
- b) Signs of pre-eclampsia are usually present.

Abdominal Examination

There are no characteristic signs.

Ultrasonography

It should be done to exclude placenta praevia. It can show a retroplacental or subchorionic haematoma in the mixed type.

Note: The bleeding is usually dark brown in colour because blood is retained for a time before its escape (prune juice discharge).

III. Mixed or Combined Haemorrhage

It is the clinical picture of concealed haemorrhage but there is vaginal bleeding.

Differential Diagnosis

1. Concealed haemorrhage: It has to be differentiated from other causes of acute abdominal pain during pregnancy or labour as rupture of uterus, red degeneration of a fibroid, acute appendicitis, etc.
2. Revealed or mixed haemorrhage: It has to be differentiated from other causes of antepartum haemorrhage: (a) placenta praevia; (b) vasa praevia; (c) local lesions in the genital tract as cervical carcinoma.

DIFFERENTIAL DIAGNOSIS BETWEEN PLACENTA PRAEVIA
AND REVEALED OR MIXED ACCIDENTAL HAEMORRHAGE

<i>Differences</i>	<i>Placenta Praevia</i>	<i>Revealed or mixed accidental haemorrhage</i>
<i>Bleeding</i>	Painless, causeless and recurrent	Bleeding is not recurrent. Accompanied by abdominal pain in the mixed type. There may be a history of trauma or pre-eclampsia.
<i>General examination</i>	– The general condition is related to the amount of blood loss because all the blood is revealed. – Signs of pre-eclampsia not necessarily present.	– General condition is related to the amount of blood loss in the revealed type but not in the mixed type. – Pre-eclampsia is usually present.
<i>Abdominal examination</i>	Page 244	There are no characteristic signs in the revealed type. In the mixed type the uterus is very tender and hard in consistency, the FHS are absent and if present they show marked fetal distress.
<i>Vaginal examination with certain precautions</i>	– Blood is usually bright red in colour. – Fullness may be felt in a vaginal fornix. – Placenta is felt in the lower segment.	– Blood is usually dark brown. – No fullness is felt. – Placenta is not felt in lower segment.
<i>Sonography</i>	Placenta is in the lower segment.	Placenta is in the upper segment.

Treatment

I. Treatment at Home

The same as mentioned before in placenta praevia.

II. Treatment at Hospital

The case is fully investigated to diagnose the cause of bleeding including ultrasound to locate the placenta.

Treatment depends upon the type of bleeding.

A) Revealed Type

1. *The patient is not in labour*

- a) If the bleeding is severe, caesarean section is performed.
- b) If the bleeding is slight or moderate, the treatment is conservative:
 - The patient remains in bed until the bleeding stops.
 - Treatment of anaemia.
 - Treatment of pre-eclampsia which is usually present.

2. *The patient is in labour*

Rupture the membranes and leave for spontaneous vaginal delivery. If bleeding continues caesarean section is done.

B) Concealed or Mixed Type

1. Antishock measures: Blood loss is replaced by fresh whole blood. A catheter is passed into the superior vena cava to monitor the central venous pressure to avoid circulatory overload (kept between 8–12 cm.water). A Foley catheter is fixed to estimate urine output. Blood is tested for the clotting factors and specific treatment is given if there is coagulation failure.
2. If the fetus is still alive an immediate caesarean section is done.
3. If the fetus is dead, oxytocin infusion is started and amniotomy is done at once to allow for vaginal delivery. However, caesarean section is done on a dead fetus if shock does not improve in spite of antishock measures. In this case the operation will evacuate the uterus and remove the cause of shock. Also the operation is done if labour fails to start within 6 hours which means that the myometrium is markedly damaged.
4. After delivery the patient is observed for:
 - a) Postpartum haemorrhage which is liable to occur.
 - b) Urine output (at least 30 ml/hour).

Note: Amniotomy reduces pain and shock (by decreasing uterine distension) and stimulates the onset of labour. It reduces the high intrauterine pressure which may lead to reflex spasm of renal vessels and renal failure (utero-renal reflex). It also decreases release of thromboplastin into the circulation.

Complications of Abruptio Placentae

I. *Maternal*

1. Shock (haemorrhagic and neurogenic).
2. Acute renal failure. Tubular or cortical necrosis may occur due to: (a) hypovolaemia leading to spasm of renal vessels and ischaemia; (b) obstruction of the glomerular

capillaries by microthrombi due to disseminated intravascular coagulation; (c) the damaged myometrium releases myoglobin which causes spasm of renal arterioles and ischaemia; (d) reflex spasm of renal vessels due to uterine distension (utero-renal reflex); (e) kidney pathology caused by pre-eclampsia; (f) incompatible blood transfusion.

3. Disseminated intravascular coagulation (DIC) and coagulation failure. The degenerated placenta produces thromboplastin which enters the maternal circulation and causes intravascular clotting. This leads to hypofibrinogenaemia, depletion of platelets and other clotting factors. Also fibrinogen and other clotting factors are consumed in the retroplacental haematoma thus aggravating the hypofibrinogenaemia. So during labour there is a continuous blood loss from the uterus due to failure of the blood to coagulate.
4. Postpartum haemorrhage because of (a) the damaged uterus may fail to contract; (b) coagulation failure due to DIC, also the fibrin degradation products inhibit myometrial contractions; (c) anaemia from blood loss leads to uterine atony.
5. Sheehan syndrome due to necrosis of the anterior lobe of the pituitary gland which complicates severe haemorrhage.
6. Ruptured uterus. It occurs with Couvelaire uterus.
7. Amniotic fluid embolism.

The maternal mortality is less than 1%. Most women die from haemorrhage or renal failure.

II. Fetal

Fetal death is due to asphyxia from premature separation of the placenta and prematurity. The fetal mortality is 30–60%.

Notes:

- Examination of Couvelaire uterus shows multiple extensive placental infarcts, retroplacental haematoma, haemorrhage in the myometrium. The peritoneal coat shows areas of ecchymosis and may be fissured leading to intraperitoneal haemorrhage. During caesarean section the uterus is stimulated to contract by giving ergometrine, oxytocin, prostaglandin, uterine massage and by compressing the uterus with hot saline packs. If atony continues hysterectomy is performed. If bleeding from the uterus is due to coagulation failure specific treatment is given (page 271).
- APH due to ruptured marginal sinus has never been supported by placental anatomists.

COMPLICATIONS OF THE THIRD STAGE OF LABOUR

1. Retention of the placenta.
2. Postpartum haemorrhage.
3. Inversion of uterus.
4. Postpartum shock.

RETENTION OF THE PLACENTA

Definition

The placenta is not delivered within half an hour after delivery of the fetus.

Incidence

0.5–1% of all deliveries.

Aetiology

1. Retention of a completely separated placenta. This is due to:
 - a) Uterine atony. The uterine contractions are weak and unable to expel the placenta.
 - b) Contraction ring (hour-glass contraction of the uterus).
 - c) Ergometrine or Syntometrine given in the active management of the third stage of labour before delivery of the placenta may cause uterine spasm and retained placenta.
2. Adherent placenta. The placenta remains attached to the uterine wall and so retained.
Two types:
 - a) Simple adhesion. The attachment of the placenta is normal and it can be removed manually. Nondetachment is due to weak uterine contraction and retraction.
 - b) Abnormal adhesion or placenta accreta.
3. The placenta may escape into the peritoneal cavity after complete rupture of uterus.
4. Abnormal adhesion of the membranes. The placenta remains suspended in the cervix or vagina by nonseparation of the membranes from the uterine wall.

Treatment

If the placenta is not delivered within half an hour after delivery of the fetus we interfere to deliver it. If haemorrhage occurs at any time, the placenta is delivered immediately disregarding the time factor.

1. The uterus is massaged to contract and ergometrine 0.5 mg or oxytocin drip is given intravenously.
2. The Brandt-Andrews method is tried to deliver the placenta.
3. If this fails, general anaesthesia is given and the method is tried once again.

4. If the second attempt fails, the placenta is removed manually. With the patient under general anaesthesia, the whole hand is introduced into the uterine cavity to remove the placenta. If there is a contraction ring halothane is given to relax the ring. If this fails dilate the ring manually to remove the placenta. In case of placenta accreta the treatment depends upon the extent of accretion. If partial, try to remove the placenta piece-meal. If the placenta is totally accreta, hysterectomy is performed. Alternative to hysterectomy in a young woman is to cut the cord short and to leave the placenta in place to undergo autolysis and spontaneous absorption. Antibiotics are given to prevent uterine infection. Some may give methotrexate to destroy the trophoblast and speed placental absorption.

Notes:

1. *Brandt-Andrews method*: The uterus is massaged to contract. The umbilical cord is fixed with one hand at the vulva while the other hand pushes the uterus upwards towards the umbilicus to separate the placenta. After separation, delivery is aided by gentle traction on the cord combined with suprapubic pressure.
2. *Crédé method*: The uterus is massaged to contract. The fundus is then squeezed between the thumb and 4 fingers to separate the placenta, then the contracted uterus is pushed downwards and backwards into the pelvis to expel the separated placenta. This method should not be used to deliver the placenta as it may lead to the following complications; (1) it may lead to pain and shock; (2) partial separation of the placenta and postpartum haemorrhage; (3) the supporting ligaments of the uterus may be stretched and lacerated leading to prolapse; (4) inversion of uterus if it is compressed while lax.
3. *Complications of retained placental fragments*: (1) Postpartum haemorrhage; (2) puerperal sepsis; (3) subinvolution of uterus; (4) placental polyp; (5) malignant trophoblastic disease.

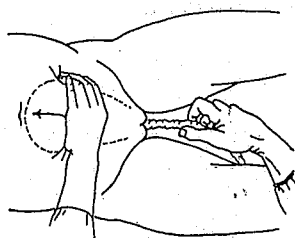


Fig.112.Brandt-Andrews method

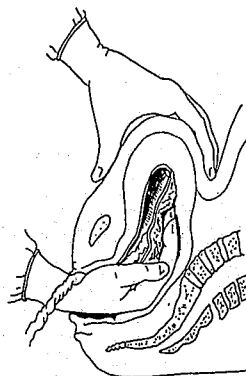


Fig.113.Manual removal of the placenta

Manual Removal of the Placenta

Under deep general anaesthesia and after evacuation of the bladder the right hand is introduced as a cone along the cord to reach the placenta and then moved to reach its lower margin. The placenta is then separated from below upwards by a side to side

movement of the hand (sawing movement) while the fundus is fixed by the other hand applied over the abdomen. After complete separation the placenta is grasped in the hand and delivered out and is inspected to detect any missing fragment. The uterus is massaged to contract and ergometrine is given I.V. Antibiotics are given to guard against infection. The complications of the procedure include: (1) Perforation of uterus; (2) retained placental fragments which may lead to the above mentioned complications.

POSTPARTUM HAEMORRHAGE (PPH)

Postpartum haemorrhage may be primary or secondary.

Primary Postpartum Haemorrhage

It is excessive bleeding from the genital tract more than 500 ml occurring during the first 24 hours after delivery of the fetus.

The normal blood loss during labour is about 300 ml and a loss of more than 500 ml usually affects the general condition of the mother and so it is considered PPH.

Secondary Postpartum Haemorrhage

It is bleeding from the genital tract occurring after 24 hours of delivery of the fetus and up to the end of puerperium (6 weeks).

PRIMARY POSTPARTUM HAEMORRHAGE

Incidence

About 5% of vaginal deliveries (1–8%). There is usually underestimation of the blood loss during labour by $1/3 - 1/2$. This is because the blood which soaks the garments, drapes, and bed linen may not be included.

Sources of Bleeding

Haemorrhage may be from the placental site or from extraplacental site due to lacerations of the genital tract, i.e. traumatic postpartum haemorrhage.

I. Placental Site Haemorrhage

Aetiology

1. Uterine atony.
2. Retention of a partially or completely separated placenta. This interferes with retraction of uterus.
3. Coagulation disorders due to disseminated intravascular coagulation, thrombocytopenia, leukemia, von Villebrand disease, etc.

Uterine Atony

It is the commonest cause of primary PPH (90%) and is due to:

1. *General causes*; as anaemia, antepartum haemorrhage, multiparity (the uterus is fibrotic), prolonged labour, excessive sedation and deep anaesthesia.
2. *Local causes*; as overdistension of the uterus by big fetus, multiple pregnancy and polyhydramnios, uterine fibroids (mechanically interfere with uterine contractions), presence of uterine scar and retention of placental fragment, blood clots, or piece of membranes.
3. *Nervous causes*; full bladder or rectum reflexly inhibits uterine contractions.
4. *Idiopathic*; no cause is found to explain the uterine atony. There may be a past history of PPH.

II. Traumatic Postpartum Haemorrhage

It is due to lacerations of the perineum, vulva, vagina, cervix or rupture of uterus. Acute inversion of uterus is a very rare cause.

Diagnosis

I. General Examination

The general condition of the patient is related to the amount of blood loss. Signs of shock occur if the bleeding is severe. Shock is manifested by rapid pulse and respiration, low blood pressure, and subnormal temperature. In fact, hypotension occurs when the blood volume is reduced by 30% or more (loss of 1500 ml or more).

II. Abdominal Examination

In atonic PPH, the uterus is large and soft, and squeezing it leads to a gush of clotted blood from the vagina. If the uterus is found hard and well contracted, the bleeding is traumatic or due to coagulation disorder.

III. Vaginal Examination

If the bleeding is bright red in colour, it suggests laceration but when the blood is dark, it indicates bleeding from the placental site. Since atony and lacerations may occur together, the perineum, vulva vagina and cervix must be inspected in every case of PPH.

IV. Examination of the Placenta and Membranes

To detect a retained placental fragment or a piece of membranes.

V. Diagnosis of Coagulopathy

Coagulopathy is suspected when there is persistent bleeding in absence of uterine atony, and lacerations in the genital tract. A coagulation profile confirms the diagnosis.

Treatment

I. Prophylactic Treatment

1. Antenatal Measures

- a) Proper antenatal care with treatment of anaemia. Haemoglobin should be kept above 11 gm% throughout pregnancy.
- b) Women predisposed to PPH as cases of multiple pregnancy, grand multipara and previous history of PPH should be delivered at hospital. These women should have their blood typed and cross-matched.

2. Intranatal Measures

- a) Proper use of analgesia and anaesthesia during labour.
- b) The routine use of ecbolics in the third stage of labour.
- c) No forceps or breech extraction before the cervix is fully dilated.

3. Postnatal Measures

- a) The placenta and membranes should be examined after delivery to exclude any missing fragment.
- b) The uterus and birth canal should be explored after any obstetric operation as forceps to detect any tear.
- c) Every patient should be observed for at least one hour after delivery to detect any PPH.

II. Active Treatment

Antishock measures including blood transfusion are started if the bleeding is severe. The next line of treatment depends upon the cause of bleeding.

Traumatic Postpartum Haemorrhage

Under general anaesthesia the birth canal is explored and tears are sutured. Sometimes a tight vaginal pack is needed to control oozing from cervix or vagina. Rupture of uterus is treated by hysterectomy or repair.

Bleeding Due to Coagulation Disorders

Specific treatment is given to correct the coagulation defect.

Bleeding Due to Retained Placenta

The placenta is delivered immediately by the Brandt-Andrews method. If this fails, do manual removal under general anaesthesia.

Bleeding Due to Uterine Atony

1. The bladder is evacuated, the uterus is massaged to contract and 0.5 mg ergometrine is given IV or IM or oxytocin is given by the intravenous drip method. If these ecbolics fail, a prostaglandin is given intramuscularly, intravenously or intramyometrial (needle passed through the abdominal wall). Also misoprostol (prostaglandin E_1 analogue) can be given rectally.
 2. If the bleeding is not controlled, the patient is given general anaesthesia and the uterine cavity is explored manually to remove retained placental tissue.
 3. If atony continues, bimanual compression of the uterus is performed. The closed fist of the right hand is placed into the anterior vaginal fornix in front of the cervix, and the left hand is placed on the abdomen behind the body of the uterus. The uterus is compressed between the two hands. In this way the placental site is compressed and bleeding is controlled. Bimanual compression is maintained until the uterus is firmly contracted. The procedure is tiring, and an assistant may be needed to replace the obstetrician after 15–20 minutes.
- If the abdominal wall is lax, the uterus is lifted out of the pelvis by one hand, while the other hand is arched to compress the lower uterine segment against the lumbar spine, thus compressing the uterine vessels (Dickinson technique).
4. If the above measures fail, abdominal hysterectomy is performed. However, if we want to keep the uterus as in the young primigravida we try the following methods:
 - a) Ligate the ascending branch of the uterine artery on both sides.
 - b) If bleeding continues, ligate the anastomosis of the uterine and ovarian artery on both sides (high at the uterine fundus just below the ovarian ligament).
 - c) If this fails, ligate the infundibulopelvic ligaments to occlude the ovarian arteries.
 - d) We can also ligate the internal iliac (hypogastric) artery or its anterior division on both sides, or do angiographic embolization of hypogastric arteries by using small pieces of Gelfoam.
 - e) A recent method is to apply the B-Lynch suture to brace the uterus to compress the bleeding sites.
 5. A tight uterovaginal pack is usually useless to control bleeding and is rarely performed.

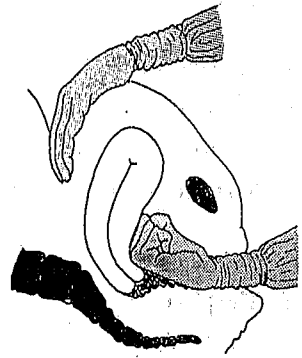


Fig. 114. Bimanual compression of uterus

Note: Ligation of both hypogastric arteries stops the bleeding in only 45% of cases due to the presence of extensive collateral circulation in the pelvis. Ligation of uterine arteries stops bleeding in 90% of cases.

Complications of PPH

- (1) Maternal death in 0-10% of cases; (2) acute renal failure; (3) Sheehan syndrome;
- (4) failure of lactation; (5) anaemia; (6) increased incidence of puerperal sepsis.

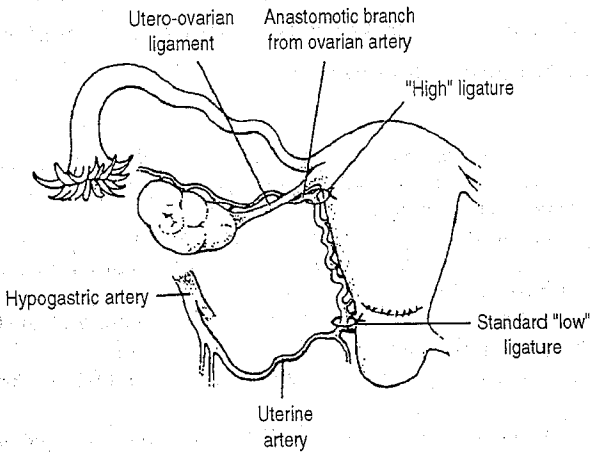


Fig. 114-2. Ligation of uterine artery.

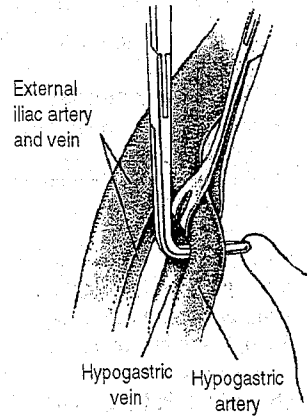


Fig. 114-3. Ligation of hypogastric artery.

SECONDARY POSTPARTUM HAEMORRHAGE

Incidence

About 0.5% of deliveries.

Aetiology

1. Retained placental tissue or piece of membranes. This is the commonest cause (40-50%). The retained tissue becomes infected and when it sloughs bleeding occurs. Also if a placental polyp forms it can cause bleeding.
2. Uterine infection. Bleeding occurs due to separation of a necrotic slough. Bleeding may come from placental site, a caesarean wound or from the cervix.
3. Subinvolution of uterus. This leads to subinvolution of placental bed and poor occlusion of spiral arteries by thrombus.
4. Submucous fibroid or fibroid polyp due to necrosis and infection.
5. Inversion of uterus.
6. Malignant trophoblastic disease (invasive mole, choriocarcinoma and placental site trophoblastic tumour).

7. Local gynaecological lesions, e.g. cervical erosion or carcinoma.
8. Haemorrhagic blood diseases as thrombocytopenia.
9. In about one-third of cases the cause is unknown.

Treatment

It is the treatment of the cause. Retained placental tissue is diagnosed by ultrasound or hysteroscope. In this case if the bleeding is slight we give ergometrine and antibiotics. However, if the bleeding persists or is severe, the uterus is curetted and the products examined histologically to exclude malignant trophoblastic disease.

Notes for the postgraduate:

1. Mechanism of haemostasis after placental separation. The uterine muscle fibres retract and compress the blood vessels in the wall of the uterus until intravascular thrombosis occurs. So uterine atony and coagulation disorders lead to bleeding.
2. Primary PPH. Uterine atony (90%), coagulation disorders (3%) and traumatic (7%).
3. Nalador. A synthetic prostaglandin. Can be given IM (500 micrograms) or by intravenous drip (4–16 micrograms/minute) to control atonic PPH.
4. Carboprost is a prostaglandin $F_{2\alpha}$ analogue, and is given as 250 micrograms IM every 15 to 90 minutes, maximum 8 doses, or 250 μ g intramyometrial.
5. Misoprostol is a prostaglandin E_1 analogue (Cytotec). It is inserted rectally in a dose of 1000 micrograms.
6. Oxytocin in PPH. 20 units in one litre normal saline or Ringer lactate solution given at a rate of 10 ml/minute.
7. Angiographic arterial embolization. Local anaesthesia. Catheter passed along femoral artery to reach aorta. A radiopaque dye is injected to show the pelvic arteries. A catheter is then passed to block the bleeding vessel or the hypogastric artery by Gelfoam (small pieces of Gelfoam; 2x2x2 mm). The material used for this technique includes gelatin sponge (Gelfoam), cellulose (Oxycell), steel coils, or silicon spheres.

INVERSION OF THE UTERUS

This means that the uterus is turned inside out. It may be acute or chronic.

Acute Inversion

It occurs during or immediately after delivery of the fetus and so it is called acute puerperal inversion.

Incidence

Rare. 1 in 2,000 to 1 in 20,000 deliveries.

Aetiology

Inversion may be spontaneous but it is usually induced.

1. Spontaneous inversion; due to precipitate labour, short cord, straining or severe coughing after labour while the uterus is lax.
2. Induced inversion; due to pressure on the fundus or traction on the cord while the uterus is lax. Placenta accreta predisposes to inversion.

Degrees

- *First degree:* The fundus is depressed so that it bulges into the uterine cavity "cupping of the fundus". The inverted fundus is still above the cervix.
- *Second degree:* The inverted fundus protrudes through the cervix into the vagina.
- *Third degree:* The fundus appears at or protrudes through the vulva.

The first and second degrees are incomplete inversion, while the third degree is called complete inversion.

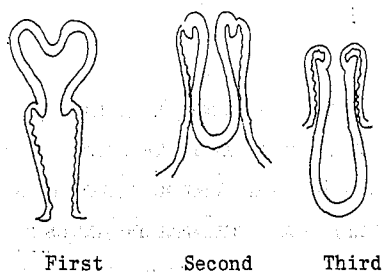


Fig. 115. Degrees of inversion

Clinical Picture

A) Symptoms

1. Pain: Severe pain may be felt in the lower abdomen. The patient cries out and bears down. Bearing down after delivery of the placenta is suggestive of inversion.
2. Haemorrhage; due to uterine atony, usually slight because the blood vessels are kinked. Bleeding is absent if the placenta remains completely attached.
3. Rarely the symptoms are mild or absent and the condition passes unnoticed until the patient develops fever and blood stained offensive vaginal discharge due to infection (subacute type).

B) Signs

1. General Examination

There is a variable degree of shock. Skin is haemorrhagic due to blood loss and neurogenic due to traction on infundibulo-pelvic ligaments and the presence of tubes and ovaries and may be intestine in the inversion cup.

2. Abdominal Examination

In the first and second degrees cupping of the fundus is felt. In the third degree the uterus is not felt per abdomen.

3. Vaginal Examination

In the second and third degrees there is a large mass in the vagina covered by dark red endometrium. The placenta may be attached to the inverted fundus.

Differential Diagnosis

1. All causes of shock after labour as rupture of uterus.
2. Fibroid polyp (by passing uterine sound).
3. Uterine prolapse (presence of external Os).

Treatment

1. Antishock measures.
2. When shock is improved, manual reduction is performed under deep general anaesthesia and halothane is added which relaxes the uterus. A tocolytic drug (as ritodrine or magnesium sulphate) can be used. Also nitroglycerin can be given IV to relax the uterus (100–200 micrograms). After reduction the uterus is massaged to contract and ergometrine is given intravenously or oxytocin drip. A broad spectrum antibiotic is given to prevent infection.
3. Reduction by the hydrostatic method (O'Sullivan) may be used. Three litres or more of sterile water may be needed.
4. When the condition is discovered some days after labour, manual reduction is tried under anaesthesia. If this fails because of tightness of the cervix, the cervical ring is divided vaginally or abdominally followed by reposition of the uterus or vaginal hysterectomy is performed if there is marked infection or the patient is old.

Prognosis

Mortality rate is 0–30%. Death is due to shock, haemorrhage, infection and pulmonary embolism.

Notes:

- Reduction by hydrostatic pressure: Done without anaesthesia. The douche nozzle is passed to reach the posterior vaginal fornix. An assistant compresses the labia against the forearm of the obstetrician to prevent escape of fluid which is allowed to run into the vagina. This leads to ballooning of the vagina and correction of inversion.
- If the placenta is still attached to the uterus, it is not removed prior to replacement of the organ to avoid bleeding from the placental area, and it is removed after reposition of the uterus. However, if the uterus cannot be replaced, the placenta has to be removed at first.

SUDDEN POSTPARTUM COLLAPSE**Aetiology**

- a) *Obstetric causes*: (1) Postpartum haemorrhage; (2) rupture of uterus; (3) extensive lacerations of cervix, vagina or perineum; (4) inversion of uterus; (5) amniotic fluid embolism; (6) eclampsia; (7) complication in an ovarian cyst as torsion or rupture.
- b) *Non-obstetric causes*: Examples include coronary thrombosis, pulmonary thrombo-embolism and anaesthetic accidents. Epidural anaesthesia may be complicated by hypotension and cardiac arrest.
- c) *Idiopathic or true obstetric shock*: This may occur after an easy and spontaneous delivery in absence of any cause which can explain the shock. Some obstetricians deny this type of shock and claim that careful examination will reveal some hidden cause as rupture of uterus or amniotic fluid embolism.

SHOCK IN OBSTETRICS

Shock is a state of circulatory failure leading to hypotension, with hypoperfusion of tissues resulting in cellular hypoxia and organs dysfunction. The blood volume in pregnancy increases by 1–2 litres and this allows the gravid woman to tolerate moderate bleeding without developing shock.

AETIOLOGY

1. *Neurogenic shock*: It occurs in painful conditions as inversion of uterus and complication in an ovarian cyst as torsion. Pain leads to vagal stimulation and this causes bradycardia and dilatation of the splanchnic vessels.
2. *Hypovolaemic shock*: It results from loss of blood (haemorrhage), plasma (burns) or water and salts (vomiting and diarrhoea). Abortion, disturbed ectopic pregnancy, hydatidiform mole, ante- and postpartum haemorrhage are causes of haemorrhagic shock. Shock may be both haemorrhagic and neurogenic as in case of placental abruption and rupture of uterus. Hyperemesis gravidarum leads to dehydration and hypotension.
3. *Cardiogenic shock*: Due to failure of the heart to pump an adequate amount of blood as in heart failure, myocardial infarction, and arrhythmias (e.g. ventricular fibrillation).
4. *Septic shock*: The new name is systemic inflammatory response syndrome. It is due to infection with Gram-negative organisms as *E. coli*, *Bacteroides*, *Proteus*, *Pseudomonas* and *Clostridium welchii*. An endotoxin present in the cell membrane of these organisms is released into the maternal circulation after bacterial death and leads to: (a) anaphylactic shock (antigen-antibody reaction); (b) dilatation of the precapillary arterioles and constriction of the postcapillary venules thus diminishing venous return; (c) disseminated intravascular coagulation (DIC); (d) direct damage of the tissues as the kidneys; (e) release of histamine from mast cells. Histamine will cause increased capillary permeability and decreased plasma volume. Septic shock may complicate septic abortion, puerperal sepsis, premature rupture of membranes, chorioamnionitis and acute pyelonephritis. Few cases of septic shock follow infection with Gram-positive organisms, fungi, protozoa or viruses. These organisms release an exotoxin.
5. *Pulmonary embolism*: Due to amniotic fluid, air or a thrombus. Amniotic fluid emboli will block the pulmonary arterioles and capillaries.
6. *Splanchnic shock*: It may follow delivery in case of multiple pregnancy and polyhydramnios. The marked pressure of the uterus on the splanchnic vessels drops after delivery leading to pooling of the blood in the splanchnic area, decreased venous return and shock.

7. *Anaesthetic shock*: Mendelson syndrome, cardiac arrest, and shock due to spinal anaesthesia.
8. *Anaphylactic shock*: It is an allergic reaction of the body to a foreign protein or drug. Hypotension results from increased capillary permeability.
9. *Endocrinal shock*: As Addisonian crisis, and hypothyroid coma.

PATHOPHYSIOLOGY

1. The decreased blood volume in hypovolaemia shock and the increased vascular bed in neurogenic shock lead to reduced venous return and reduced cardiac output.
2. The sympathetic nervous system becomes activated leading to tachycardia and vasoconstriction of the peripheral arterioles. Due to decreased intravascular hydrostatic pressure, fluid moves from the interstitial space into the vascular space to restore the blood volume. This gives the sensation of thirst. However, if the blood loss is severe (blood volume is reduced by 30% or more, i.e., loss of 1500 ml or more) these haemodynamic changes fail and the blood pressure drops.
3. The blood flow goes mainly to the brain and heart at the expense of other organs. Diminished blood supply to the skin makes it pale, cold, clammy with cyanosis of the fingers. Reduced blood flow to the kidneys leads to oliguria and anuria.
4. Tissue hypoxia leads to anaerobic metabolism and metabolic acidosis. Acidosis makes respiration shallow and rapid (Kaussmaul respiration).
5. Hypoxia disturbs the function of the cell membrane leading to influx of sodium and water and loss of potassium (hyponatraemia and hyperkalaemia).
6. Tissue damage by hypoxia releases thromboplastin which causes DIC.
7. Finally the coronary and cerebral blood flows are reduced leading to heart failure, coma and death.

CLINICAL PICTURE

The main features of shock are:

1. Weak rapid pulse (above 100 beats/minute). Bradycardia occurs if there is cardiac pathology or vagal stimulation in neurogenic shock.
2. Low blood pressure (systolic below 90 mmHg or a drop of 40 mmHg or more if hypertension was present).
3. Subnormal temperature.
4. Shallow rapid respiration.
5. Skin is pale, cold and clammy with cyanosis of fingers.
6. Oliguria or anuria. Anuria occurs if the systolic pressure falls to 80 mmHg or less.

TREATMENT

A. General Measures

1. The legs of the patient are raised by 30 degrees to increase venous return and cardiac output. Head-down position may lead to difficult breathing in case of abdominal or chest trauma.
2. Oxygen is given by nasal catheter, mask, or endotracheal tube if patient is comatosed.
3. Patient is kept warm with blanket. Excessive heat is avoided (this will cause vasodilatation and in fact peripheral vasoconstriction is a protective mechanism to maintain the blood supply to the vital organs).
4. Morphine 10–15 mg intravenously, if there is pain or the patient is irritable (if given IM, the absorption will be poor because of peripheral vasoconstriction).

B. Fluid Replacement

The patient is supplied with the same type of fluid she has lost. Until blood is available, the patient is given a crystalloid solution as 5% dextrose, normal saline, Ringer lactate or Hartman solution. Also plasma and plasma substitutes can be given. Measuring the central venous pressure (CVP) or pulmonary capillary wedge pressure (PCWP) allows adequate fluid replacement without overloading the circulation.

C. Drugs

1. Vasoactive (Inotropic) Drugs

Given if shock does not respond to fluid replacement.

- a) Cardiotonic drugs as dopamine. This drug improves myocardial contractility and selectively improves cerebral, coronary, renal and mesenteric blood flow.
- b) Corticosteroids. They elevate the blood pressure by increasing the sensitivity of blood vessels to circulating catecholamines and draw fluids into the vascular space (due to salt retention).

2. Antibiotics

Given in large doses intravenously in case of septic shock.

3. Sodium Bicarbonate

Given intravenously to correct metabolic acidosis (pH is below 7.2. Dose is 100 mEq I.V.).

D. Monitoring of the Patient

1. Observation of the vital signs; pulse, temperature and blood pressure.

2. Fluid intake and urine output measured via a Foley catheter. Urine output should not be less than 30 ml/hour. If less, it is due to poor renal perfusion (concentrated urine and specific gravity above 1020) or renal failure (specific gravity below 1010).
3. Blood tests: (a) Complete blood count. The haematocrit value starts to drop when fluid migrates from interstitial space into the vascular space. We aim to keep it 30% or above which is optimal for oxygen transfer; (b) blood urea nitrogen and serum creatinine to assess kidney function; (c) serum electrolytes (sodium, chloride, potassium, calcium and bicarbonate) to select the type of fluid to be given; (d) arterial blood gases to know acid-base status; (e) coagulation profile for early detection of DIC which is a common finding; (e) chest X-ray and electrocardiogram.

Notes for the Postgraduate

- In severe vasoconstriction or when systolic pressure is below 55 mmHg the sphygmomanometric reading becomes inaccurate or unobtainable and direct intra-arterial readings should be obtained via an arterial line.
- Reduction of the blood volume by 10–20% (mild shock), 20–40% (moderate shock), more than 40% (severe shock).
- CVP. It is measured by passing a catheter along a vein in the elbow or external jugular vein to reach the superior vena cava. The aim is to keep the pressure between 8–12 cm. water.
- PCWP. The Swan-Ganz catheter is passed into the pulmonary artery to measure the PCWP which reflects the left atrial pressure and the function of the left heart. It is a more complicated procedure but more sensitive than CVP in detecting fluid overloading and pulmonary oedema. Indicated when monitoring of the left heart function is essential. The normal value is 6–12 mmHg.

COAGULATION DEFECTS IN OBSTETRICS

Deficiency of coagulation factors with tendency to bleeding results from disseminated intravascular coagulation (DIC), dilutional coagulopathy and excessive infusion with dextran solution.

I. DISSEMINATED INTRAVASCULAR COAGULATION

When DIC occurs there is consumption of blood platelets, fibrinogen and other clotting factors. This is accompanied by increased activity of the fibrinolytic system to produce lysis of the fibrin thrombi to maintain patency of the microcirculation. This results in increased fibrin degradation (split) products. Bleeding occurs when plasma fibrinogen reaches 100 mg% or less. In this case, the blood fails to coagulate. During pregnancy the fibrinogen level is 400-600 mg%.

Aetiology

The causes of DIC and hypofibrinogenaemia in obstetrics include:

1. Severe placental abruption (concealed type). Commonest cause and responsible for 60-70% of cases (page 254).
2. Missed abortion. The degenerated placenta or dead fetus liberates thromboplastin which enters the maternal circulation causing intravascular coagulation (it converts prothrombin into thrombin).
3. Intrauterine death.
4. Septic abortion, puerperal sepsis, chorioamnionitis and acute pyelonephritis. The endotoxin of Gram-negative organisms as *E. coli* leads to; (a) damage of vascular endothelium; (b) clumping of platelets; and (c) activates factor 12.
5. Amniotic fluid embolism. The amniotic fluid contains thromboplastin.
6. Severe pre-eclampsia and eclampsia. Due to damage of capillary endothelial cells and release of thromboplastin from degenerated placenta. Also, antithrombin III may be decreased secondary to severe proteinuria.
7. Uterine rupture and any extensive tissue damage; (a) trauma may cause release of thromboplastin from damaged tissues in the circulation; (b) formation of a large haematoma may consume fibrinogen and other clotting factors; (c) uterine rupture may allow amniotic fluid to enter maternal circulation and this acts as a source of thromboplastin.
8. Incompatible blood transfusion. Red cell haemolysis leads to release of thromboplastin and adenosine diphosphate which causes aggregation of platelets.
9. Acute fatty liver of pregnancy. It may present like pre-eclampsia, but there is also nausea, vomiting and liver dysfunction.

10. Induction of abortion with intra-amniotic injection of hypertonic saline or urea solution. This will cause necrosis of the placenta, decidua and fetal tissues with release of thromboplastin from degenerated tissues.

Diagnosis

- a) Failure of the blood to coagulate may give rise to epistaxis, haematuria, bleeding from needle puncture sites and postpartum haemorrhage. The patient will show the clinical picture of the underlying cause as abruptio placentae and intrauterine death.
- b) The clot observation test (Weiner test). It is a rapid bedside test. Five ml of venous blood are withdrawn in a test tube. Failure of any clot to form within 10 minutes indicates that fibrinogen is less than 100 mg%. If a clot forms the tube is incubated at 37° C (body temperature). If the clot dissolves after 30 minutes it means excessive fibrinolytic activity.
- c) A coagulation profile is ordered which will show:
 1. Plasma fibrinogen is reduced.
 2. Fibrin degradation (split) products are increased (more than 40 micrograms/ml).
 3. Platelet count is decreased (<150,000 per cu.mm).
 4. Prothrombin time (PT) is increased (N: 10–15 seconds).
 5. Thrombin time (TT) is increased (N: 25–35 seconds).
 6. Partial thromboplastin time (PTT) is increased (N: 25–35 seconds).
 7. Antithrombin III consumption is increased so its level or activity is decreased (N: 22–39 mg/dl or activity 84–123%). This factor prevents formation of thrombin.
 8. D-Dimers. These products rise when there is thrombosis and are more specific than fibrin degradation products (plasma level above 0.5 microgram/ml is abnormal).

Treatment

1. Treatment of the cause, e.g., if DIC is due to infection, broad spectrum antibiotics are given.
2. Fresh whole blood to replace the blood volume, red cells and clotting factors. If whole blood is not available, give packed red cells and fresh frozen plasma (FFP) or cryoprecipitate (one litre of FFP supplies 3 grams of fibrinogen and all the clotting factors).
3. Platelets are transfused if platelet count is below 50,000/cu.mm.
4. Administration of antithrombin III concentrate may improve the condition.
5. Heparin. In case of a dead fetus and when laboratory tests indicate DIC but the patient is not bleeding (intact vascular tree) heparin is given to raise the fibrinogen level to above 200 mg%. Heparin is then stopped and labour is induced after 6 hours.

Note: The coagulation defect corrects itself spontaneously within 24 hours after delivery or removal of the cause.

II. DILUTIONAL COAGULOPATHY

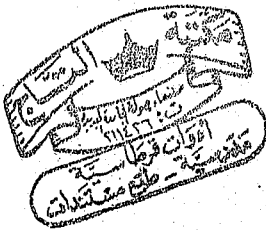
This can result if large amounts of intravenous fluids or blood (5 units or more) are given. This leads to dilution of coagulation factors and tendency to bleeding. So a unit of FFP should be given for every 5 units of transfused blood.

III. DEXTRAN INFUSION

Excessive infusion with dextran (more than one litre) may cause coagulopathy. Dextran forms a fibrinogen-dextran complex leading to hypofibrinogenaemia, and prevents aggregation of platelets.

Notes:

- Fibrin degradation products prevent formation of fibrin clots, inhibit myometrial contractions and cause toxic myocarditis. So they increase bleeding and shock.
- Cryoprecipitate. Obtained from fresh frozen plasma by warming to 4°C. It supplies fibrinogen and other clotting factors. Each unit of cryoprecipitate raises the fibrinogen level about 10 mg/dl.
- One unit of platelets raises the platelet count by 10,000/mm³.
- Causes of hypofibrinogenaemia include causes of DIC, dilutional coagulopathy and dextran infusion.
- Dextran interferes with cross-matching.



AMNIOTIC FLUID EMBOLISM (AFE) **(Anaphylactoid Syndrome of Pregnancy)**

INCIDENCE

1 in 8000 to 1 in 80,000 deliveries. The latter figure is nearer to the true incidence.

AETIOLOGY

The exact cause is unknown. It may be due to sudden escape of a large volume of amniotic fluid into the maternal venous circulation after rupture of membranes. The amniotic fluid emboli will block the pulmonary arterioles and capillaries leading to pulmonary hypertension and hypoxia. Hypoxia leads to left ventricular heart failure and death. The condition tends to occur in labours with strong uterine contractions and when the uterus is excessively stimulated by oxytocin infusion or prostaglandin. It may also occur during caesarean section and after rupture of the uterus.

Recently, it is suggested that AFE is an immune response caused by exposure of the mother to amniotic fluid leading to anaphylactic reaction and shock. It usually occurs at the time of delivery, but has been reported after amniocentesis and therapeutic abortion. Also uterine hyperstimulation is found in less than 10% of patients.

CLINICAL PICTURE

The patient develops sudden severe dyspnoea, cyanosis and cough with frothy, blood-stained sputum. This is immediately followed by apnoea and profound shock. Chest pain is unusual. Death occurs in 25% of cases within one hour and the majority who survive will develop disseminated intravascular coagulation (DIC) with bleeding from the genital tract and from all sites of trauma.

DIAGNOSIS

It is based on clinical picture and laboratory studies which include (a) complete blood count, (b) arterial blood gases, and (c) coagulation profile.

Diagnosis in the past was confirmed by postmortem examination to detect the presence of amniotic fluid elements as fetal squamous cells (from skin), lanugo hair, vernix caseosa, and meconium in pulmonary arterioles and capillaries. However, only 50-70% of patients dying from this syndrome have this finding. Also amniotic fluid elements in the pulmonary circulation are detected in normal pregnancy.

DIFFERENTIAL DIAGNOSIS

This includes pulmonary thromboembolism, aspiration pneumonia, myocardial infarction, and septic shock.

TREATMENT

1. Oxygen is supplied by an endotracheal tube and mechanical ventilation.
2. Treatment of shock:
 - a) Blood volume expansion by colloids, crystalloids, or blood.
 - b) Pressor agents such as dopamine or norepinephrine.
3. Digitalis if there is left ventricular failure which is common.
4. Treatment of pulmonary oedema which is common (70%) by diuretics as frusemide (Lasix).
5. Corticosteroids may help as the syndrome may be immune mediated (hydrocortisone 500 mg IV every 6 hours until the patient recovers).
6. When DIC occurs, it is treated properly.
7. If maternal cardiac arrest occurs before delivery, the fetus is delivered immediately by forceps or by caesarean section if conditions are not favourable for vaginal delivery.

PROGNOSIS

Maternal mortality was 85%, but now it is about 60%. Many of the women (75%) who survive will suffer neurological damage secondary to hypoxia. Neonatal survival is about 75% with 50% of those surviving will suffer neurological problems.

BLOOD TRANSFUSION IN OBSTETRICS

INDICATIONS

1. Haemorrhage. Haemorrhage causing a lowering of systolic blood pressure below 90 mmHg and a heart rate above 100 beats/minute indicates a good amount of blood loss (more than 1500 ml) and the need for transfusion.
2. Shock. Plasma is preferred if shock is not associated with haemorrhage.
3. Severe anaemia near term (after 36th week) for rapid correction before onset of labour.
4. Septic abortion and puerperal sepsis. Fresh blood helps to control infection.
5. Erythroblastosis fetalis. Blood may be needed for intrauterine transfusion or exchange transfusion after delivery of the fetus.
6. Haemorrhagic disease of the newborn.

COMPLICATIONS

1. Haemolytic reactions. Excessive haemolysis of the donor's cells may occur due to transfusion of incompatible blood, old stored blood or overheated blood. The condition is manifested by rise of temperature, rigors, dyspnoea, cyanosis, headache, pain in the chest and lumbar region. Later anuria and jaundice may occur. It is advisable to start the blood transfusion rapidly and not slowly so that any reaction will appear early before a large amount of blood is given.
2. Febrile reactions; due to the presence of pyrogens (products of bacterial metabolism) in the blood or in the apparatus.
3. Allergic reactions as urticaria due to antigens in the transfused plasma.
4. Septicaemia because of infected blood. If blood is kept outside the refrigerator more than 30 minutes it should not be used, as the room temperature allows multiplication of bacteria.
5. Transmission of diseases as malaria, syphilis, viral hepatitis and AIDS (acquired immune deficiency syndrome).
6. Air embolism.
7. Thrombophlebitis of the vein used for transfusion.
8. Rh sensitization.
9. Overtransfusion and heart failure.
10. Citrate intoxication if large amounts of citrated blood are given rapidly (1500 ml in 15 minutes or 5000 ml in 2 hours). The effects are a fall in blood pressure, tremors, convulsions and cardiac arrhythmias. It is treated by 1 gram calcium I.V. (10 ml 10% solution).
11. Potassium intoxication. This occurs with stored blood. On storage potassium diffuses from red cells to the plasma and this increases with time.
12. Dilutional coagulopathy (page 272).

PUERPERIUM

NORMAL PUERPERIUM

Puerperium is the period after delivery during which the genital organs involute, i.e. return to their condition before pregnancy. The duration is 6 weeks.

PHYSIOLOGICAL CHANGES OCCURRING DURING THE PUERPERIUM

A) General Changes

1. *Pulse*: It is normal, may be accelerated if there is haemorrhage or infection.
2. *Temperature*: It is normal, but usually there is a slight rise during the first day, it is known as "reactionary rise". It does not exceed 38° C and is not accompanied by increased pulse rate. This low-grade fever may be due to dehydration, or transient bacteraemia. It will resolve spontaneously in the majority of cases.
3. *Blood*: All the changes in the blood that had occurred during pregnancy gradually disappear during puerperium. The increased coagulability of the blood continues for 2 weeks after delivery due to decreased plasma volume.
4. *Breast*: Colostrum is secreted during the first 3 days. Breast engorgement frequently occurs on the 3rd or 4th day of puerperium when milk secretion starts.
5. *Bowels*: There is usually constipation and slight abdominal distension. Constipation is due to dehydration, anorexia and low oral intake, atony of the intestine and abdominal wall, and sometimes reflex inhibition of defecation by a painful episiotomy wound.
6. *Body weight*: Slight loss in the first 2 days due to evacuation of uterine contents, polyuria and excessive sweating.
7. *Urine*: There is diuresis especially in the first two days. Lactosuria is present during the early phase of lactation. Small amounts of peptones are present from the second to the tenth day (autolysis of uterine muscle fibres).
8. *Skin*: There is tendency to excessive sweating.
9. *Postnatal blues*. These occur in more than 50% of postpartum women. See postnatal care.
10. *Menstruation*. It starts 6–8 weeks after delivery if the patient is not lactating. With lactation there will be a variable period of amenorrhoea.

B) Local Changes

1. Uterus

a) Size

Immediately after delivery the fundus is at the level of the umbilicus, after one week, it is midway between the umbilicus and symphysis pubis. At the end of the second week it is at the upper border of symphysis pubis and it gradually diminishes in size.

b) Weight

Immediately after delivery, its weight is about 1000 g. After one week it is 500 g, at the end of the second week it is 250 g and it gradually diminishes in weight to become about 50 to 70 g.

c) Structure

- i) The cytoplasm of muscle fibres undergo autolysis by action of enzymes and each muscle fibre becomes shorter and thinner. Peptones appear in urine.
- ii) The blood vessels in the wall of the uterus are obliterated by thrombosis and replaced by smaller ones. Remnants of the old vessels can be detected as elastic fibres.
- iii) The decidua except the basal layer is shed and discharged in the lochia. The endometrium regenerates from the left basal layer.

2. The Lochia

It is the discharge escaping from the vagina during the puerperium. It arises from the raw placental area. Duration 3–6 weeks. It is red in colour for the first 5 days (lochia rubra), then it becomes yellow for 5 days (lochia serosa) then it becomes white (lochia alba) and disappears. The lochia is alkaline in reaction and contains decidual fragments, blood, cervical mucus, vaginal epithelial cells and bacteria. The lochia which remains red in colour and excessive in amount indicates delayed involution which may be associated with retained placental tissue or infection. Offensive lochia suggests but does not prove infection.

3. Cervix, Vagina and Vulva

- i) The cervix is closed by the end of the first week.
- ii) Gaping of the vulva, laxity of the pelvic floor and vaginal walls disappear by the end of puerperium.

4. The cervical ligaments involute, subinvolution leads to uterine prolapse and retroversion.

SUBINVOLUTION OF UTERUS

It is failure of the uterus to return to its original nonpregnant size within 6 weeks after delivery. The causes are (1) infection; uterine or extrauterine; (2) retained placental tissue or piece of membranes; (3) fibroids; (4) overdistension of the uterus, as in cases of polyhydramnios and twins; (5) retroversion-flexion of the uterus and congestion; (6) bad general condition due to anaemia, antepartum or postpartum haemorrhage; (7) thick decidua difficult to undergo autolysis; (8) multiparity; (9) nonsuckling (suckling stimulates the posterior pituitary to release oxytocin which causes uterine contraction and involution).

The symptoms may be in the form of persistence of lochia, continuous or irregular uterine bleeding, backache and a sensation of weight in the pelvis. On examination the uterus is larger and softer than normal for the given postpartum period. Treated by ergometrine tablets or drops given for 5 days. If bleeding is excessive or persistent the uterus is curetted to remove any retained placental fragments.

PUERPERAL SEPSIS

It is bacterial infection of the genital tract occurring after delivery.

AETIOLOGY

1. Causative Organisms

Any pathogenic organism (aerobic and anaerobic) can cause puerperal sepsis. The organisms include staphylococci, streptococci, *Escherichia (E.) coli*, *Clostridium welchii* as well as other organisms. Mixed infection may occur. The anaerobic streptococci are the commonest organisms. However, in the majority of cases the infection is polymicrobial.

2. Modes of Infection

- a) *Exogenous*: The organisms are transmitted to the genital tract from an external source such as droplet infection, dust, flies or instruments.
- b) *Endogenous*: It is infection by organisms already present in the genital tract, e.g. anaerobic streptococci.
- c) *Autogenous*: The organisms are transmitted to the genital tract from a septic focus in the patient. This occurs by the bloodstream or by the fingers of the patient.

3. Predisposing Factors

- a) *General causes*: As anaemia, diabetes, malnutrition, antepartum and postpartum haemorrhage and prolonged labour.

- b) *Local causes*: As premature rupture of the membranes, lacerations, retained placental fragments, pieces of membranes, blood clots and intrauterine manipulations.

SITES OF INFECTION

1. *Primary sites*: These include: (a) lacerations in the genital tract; (b) the placental site; (c) tissues retained in the uterus as placental fragments, pieces of membranes or blood clots.
2. *Secondary sites*: Infection may spread from the primary sites directly, by lymphatics or veins to affect the tubes, ovaries, pelvic peritoneum, parametrium or pelvic veins. Spread by the bloodstream leads to septicaemia or pyaemia.

PATHOLOGY

Infection in the uterus occurs in two forms:

1. Localized or Putrid Endometritis

The infection is mild and resistance of the patient is high. Infection is limited to the superficial layer of the endometrium by a well formed zone of leucocytic infiltration. The uterus is large, soft and contains placental fragments, pieces of membranes or blood clots. The lochia is excessive and offensive.

2. Spreading or Septic Endometritis

The infection is severe and resistance of the patient is low. The uterus is of normal size, it does not contain placental fragments. The cavity is lined by a yellow purulent membrane. Microscopically the leucocytic zone or barrier is absent or defective allowing spread of infection beyond the uterus. The lochia is scanty and not offensive.

Note: The endometrium is the commonest site of puerperal sepsis.

CLINICAL PICTURE

This depends upon the site of infection:

1. *Infected tears* cause local pain and mild fever, sometimes dysuria and retention of urine.
2. *Puerperal endometritis*: Fever usually occurs on the third day. There is lower abdominal pain. The uterus is tender and the lochia is usually excessive and offensive.
3. *Puerperal septicaemia*: Onset is usually on the third or fourth day, temperature is high and hectic. The pulse is rapid but it is out of proportion to the rise of temperature. Severe headache, vomiting and repeated rigors. The lochia is scanty and not offensive.
4. *Salpingo-oophoritis*: There is a sudden onset of severe lower abdominal pain bilateral in nature together with fever, rigors and vomiting. The lower abdomen is rigid and tender

with maximal tenderness at the tubal points. Vaginally, the lateral fornices are tender and severe pain occurs when the cervix is moved from side to side.

5. *Parametritis*: It occurs in the second week. There is fever, rapid pulse, lower abdominal pain and backache. On bimanual examination there is a hard tender mass to one side of the uterus and reaching the lateral pelvic wall. It may be bilateral.
6. *Peritonitis*: Pelvic peritonitis causes fever, rapid pulse, repeated vomiting, lower abdominal pain, tenderness and rigidity. The Douglas pouch is full and tender. In general peritonitis the symptoms are more severe and there is generalized abdominal pain, tenderness and rigidity.
7. *Pelvic thrombophlebitis*: It starts in the second week of puerperium. There is mild fever, pelvic pain and chills may occur. If the condition is neglected it may lead to pulmonary embolism, pyaemia, or thrombosis of inferior vena cava. The condition is suspected in a patient responding poorly to antibiotic therapy for endometritis. Diagnosis is confirmed by Doppler ultrasound, CT scan, or MRI.

TREATMENT

I. Prophylactic Treatment

A) During Pregnancy

Treatment of anaemia, diabetes mellitus, and septic foci. Avoid sexual intercourse during the last month.

B) During Labour

1. Aseptic and antiseptic measures should be followed.
2. Avoid unnecessary vaginal examination.
3. Prophylactic antibiotics if labour is prolonged more than 24 hours or if there is premature rupture of membranes more than 18 hours or instrumental delivery.

C) During the Puerperium

1. Infected persons or carriers should not come in contact with the puerpera.
2. Maintain aseptic and antiseptic measures.
3. Early isolation of infected cases.

II. Actual Treatment

A) General Measures

1. Isolation in a separate room.
2. Semisitting position to help drainage.
3. A light diet and plenty of fluids.

4. Correction of anaemia.
5. Symptomatic treatment as analgesics for pain and antipyretics.

B) Antibiotics and Chemotherapy

A cervicovaginal swab is taken from cervical canal and vaginal vault for culture (aerobic and anaerobic) and sensitivity test. In the majority of cases infection is polymicrobial with mixed aerobic and anaerobic organisms. A triple antibiotic therapy is given including penicillin (or ampicillin), gentamycin, and clindamycin (or metronidazole). After 48 hrs, either continue or shift to another drug according to the clinical response and the sensitivity test. In *Clostridium welchii* infection antitoxin serum is also given.

C) Drainage

1. Fowler or semisitting position. It helps drainage from the birth canal.
2. Infected tears: The sutures are removed to help drainage.
3. Pelvic abscess is drained through a posterior colpotomy.
4. If a placental fragment is blocking the cervix, it is removed by the ovum or ring forceps.

D) *Treatment of special forms of infection* as thrombophlebitis and peritonitis. Thrombophlebitis is treated by antibiotics and anticoagulation with heparin for 7 to 10 days. The antibiotic therapy must cover aerobic and anaerobic organisms.

PUERPERAL PYREXIA

DEFINITION

It is a rise of temperature reaching 38° C or higher occurring on two or more occasions during the first 10 days after delivery excluding the first 24 hours. The temperature has to be taken by mouth 4 times daily (6 hourly).

CAUSES

1. Puerperal sepsis.
2. Breast complications as acute mastitis.
3. Urinary tract infection as cystitis or pyelonephritis.
4. Intercurrent infection as tonsillitis, typhoid or malaria, etc.
5. Complicated pelvic tumour as infected ovarian cyst or red degeneration in a fibroid.

DIAGNOSIS

Every case of puerperal pyrexia is considered a case of puerperal sepsis until proved otherwise.



A) History

1. History of previous infection as pulmonary tuberculosis or urinary tract infection.
2. Obstetric history: The nature of delivery in detail; spontaneous or instrumental, duration of labour, time of rupture of membranes and catheterization of the bladder.
3. Present history: Time of onset of fever. The presence of other symptoms as sore throat, pain in the breast, abdominal pain or painful micturition, etc.

B) General Examination

1. Pulse and temperature. In septicaemia the pulse rate is out of proportion to the rise of temperature.
2. The presence of anaemia, jaundice or skin rash (septicaemia).
3. Mouth examination for tonsillitis, sore throat or septic teeth.
4. Examination of the chest; heart (endocarditis), lung (pneumonia) and breasts (mastitis).
5. Lower limbs for venous thrombosis.

C) Abdominal Examination

The height of the fundus, the presence of tenderness, rigidity or abdominal mass.

D) Vaginal Examination

1. Note the amount, colour and odour of the lochia.
2. The vulva and perineum are inspected for infected lacerations.
3. Bimanual examination to note the size of the uterus and to detect pelvic swellings.
4. Speculum examination of the cervix and vagina.

E) Special Investigations

1. Complete blood count.
2. Blood culture (aerobic and anaerobic) if there is evidence of septicaemia or temperature reaching 39° C.
3. Urine analysis, culture and sensitivity test.
4. A cervicovaginal swab is taken from the cervical canal and vaginal vault. Cultures (aerobic and anaerobic) and sensitivity tests are performed.
5. Other investigations may be required as X-ray to the chest, Widal test or a blood film for malaria.

BREAST DISORDERS IN THE PUERPERIUM**1. CRACKED NIPPLES**

Cracked nipples cause pain during suckling. There is also a risk of a mammary abscess forming, as the ducts are not emptied because of pain, also the cracked area allows entry of

infecting organisms. A nipple shield is used during feeding to prevent further injury. Panthenol or similar cream is applied during the day and compound tincture of benzoin is applied at night. If these measures fail the infant should not be given the affected breast which must be emptied regularly by manual compression or by a pump until the lesion is completely healed.

2. BREAST ENGORGEMENT

It frequently occurs on the third or fourth day after delivery when milk secretion starts. It is due to obstruction of the mammary ducts with coagulated milk or absence of the milk ejection reflex and so the alveoli become distended with milk. The distended alveoli obstruct the lymphatics and veins in between and so the breasts become enlarged, engorged, painful and tender. There is malaise and moderate rise of temperature due to absorption of milk proteins. The temperature rarely exceeds 38.5°C and does not last more than 24 hours.

Treatment

1. Support the breasts with a binder or brassiere.
2. Analgesics.
3. Ice bag to relieve pain and congestion.
4. Evacuation of the breast by manual compression or a pump.
5. Oxytocin can be given as nasal spray or sublingually at the time of feeding to help milk flow.

3. ACUTE MASTITIS AND BREAST ABSCESS

Causative Organisms

The commonest organism is the staphylococcus aureus, however, infection can be caused by other organisms as streptococci, E. coli, etc. The infant nose or throat is the source of infection (the infant becomes infected as he comes in contact with infected persons). The organism enters the nipple at the site of a fissure or abrasion at the time of breast feeding.

Forms of Mastitis

1. Infection may be limited to the areola forming a subareolar abscess.
2. Infection spreading along the duct system causes parenchymatous mastitis.
3. Infection passing along lymphatics leading to interstitial mastitis.
4. Infection may involve the areolar tissue under the breast producing a retro-mammary abscess.
5. Rarely a subcutaneous abscess forms.

Clinical Picture

The patient complains of pain in the breast with fever, headache and chills. The breast is enlarged, tender, warm and red. The axillary glands are usually enlarged and tender. If an abscess forms, there is oedema of the skin and fluctuation.

Treatment

- A) *Mastitis*: (1) Antibiotics are started after taking a sample of milk for culture and sensitivity test; (2) analgesics; (3) hot fomentations to relieve pain and reduce the inflammation; (4) support of the breast with a binder or a brassiere; (5) breast feeding from the affected breast is suspended and the breast is emptied by manual expression or with a pump.
- B) *Breast abscess*: (1) Incision and drainage under general anaesthesia; (2) a sample of pus is taken for culture and sensitivity test; (3) antibiotics; (4) stop lactation.

4. GALACTOCELE

The lumen of a large mammary duct is obstructed by inspissated secretion or inflammatory changes in its wall forming a retention cyst containing milk. It forms a local fluctuating swelling. It may resolve spontaneously or requires aspiration.

5. CARCINOMA OF THE LACTATING BREAST

When carcinoma develops in the nursing breast, it grows rapidly and is very malignant. Absence of pain differentiates it from an abscess.

6. DEFICIENT LACTATION

Aetiology

1. Failure of the baby to suckle efficiently.
2. Ill health and poor nutrition.
3. Emotional states.
4. When labour is complicated by shock, haemorrhage or infection.
5. Destruction of the anterior lobe of the pituitary gland "Sheehan syndrome".
6. Elderly primipara.
7. Poor development of the gland tissue of the breast.
8. Destruction of the gland tissue by previous mastitis.

Management

1. Excessive fluids, at least 3 litres daily.
2. Proper diet.
3. Regular breast feeding.

4. The baby should suckle from both breasts.
5. If the baby fails to empty the breast, any remaining milk should be removed by pump or manual expression.
6. Prolactin preparations may help.

CONTRAINDICATIONS TO BREAST FEEDING

I. Maternal Conditions

1. *Maternal diseases:* (a) Heart failure; (b) active pulmonary tuberculosis; (c) chronic renal disease; (d) acute illness as pneumonia.
2. *Diseases of the breast:* (a) mastitis and abscess; (b) marked fissuring or retraction of the nipples.

II. Fetal Conditions

1. Marked prematurity.
2. Congenital malformations which interfere with suckling as harelip and cleft palate.

Inhibition of Lactation

This is achieved by: (1) A dopamine agonist is given. Lisuride (Dopergin) one tablet (0.2 mg) twice daily for two weeks; (2) vitamin B6 tablets, 100 mg three times daily for 5 days. Pyridoxine shares in the synthesis of dopamine which is the prolactin inhibitory hormone; (3) clomiphene (Clomid) 2 tablets daily for 5 days; (4) diuretics; (5) tight breast binder to prevent accumulation of milk.

Notes:

- Oestrogen predisposes to venous thrombosis and is no more used to inhibit lactation.
- Myocardial infarction and cerebrovascular accidents were reported in some women who received bromocriptine (Parlodel) to inhibit lactation, and so the drug is not used for this purpose.

MATERNAL MORTALITY

The maternal mortality ratio or rate is the number of maternal deaths resulting from pregnancy, labour or puerperium per 100,000 live births. It is 8 per 100,000 live births in United States and 83 per 100,000 live births in Egypt. The maternal mortality ratio (MMR) is calculated as follows:

$$\text{MMR} = \frac{\text{Number of maternal deaths in one year}}{\text{Number of live births in the same year}} \times 100,000$$

The maternal mortality includes both direct and indirect maternal deaths.

- *Direct maternal death.* This includes death of the mother resulting from obstetrical complications of pregnancy, labour, or the puerperium (first 6 weeks after delivery). An example is maternal death resulting from rupture of the uterus.
- *Indirect maternal death.* This includes death of the mother resulting from a disease which is aggravated by pregnancy. An example is maternal death resulting from heart disease.

FACTORS WHICH AFFECT MATERNAL MORTALITY

1. *Age:* The safest age for childbearing is from 20 to 24 years. Teenage pregnancy (before 20 years) is associated with a higher MM, because women become fertile several years before their bodies are able to cope with pregnancy and childbirth. Also age above 30 years is associated with higher MM.
2. *Parity:* Grand multiparity (5 or more deliveries) is associated with a higher MM irrespective of age. The second and third deliveries are associated with least maternal complications and death increases steadily thereafter.
3. *Lack of adequate spacing between pregnancies* increases the risk to the mother because she has no time to recover from the strain of pregnancy and lactation. An interval of 2 to 3 years between pregnancies seems to be ideal.
4. *Mode of delivery:* Spontaneous vaginal delivery is safer compared to instrumental or caesarean deliveries.
5. *Place of delivery:* Home deliveries in developing countries are associated with a higher MM. High risk patients should deliver at hospital.
6. *Education of the mother.* It has an influence on MM.
7. *The use of contraceptives.* To avoid the risks of unsafe termination of pregnancy as haemorrhage and infection.

AETIOLOGY

1. Shock as in rupture or inversion of uterus.
2. Haemorrhage. Abortion, disturbed ectopic pregnancy, hydatidiform mole, ante and postpartum haemorrhage, etc.
3. Infection. Septic abortion and puerperal sepsis.
4. Hypertensive disorders of pregnancy.
5. Thrombosis and pulmonary embolism.
6. Amniotic fluid embolism.
7. Heart disease and other diseases which become aggravated by pregnancy.
8. Complications of anaesthesia.

The commonest causes in sequence are pulmonary thromboembolism, hypertensive disorders of pregnancy, haemorrhage (mainly postpartum), amniotic fluid embolism, and infection. This applies to developed countries. In developing countries the main cause of maternal death is haemorrhage.

MEASURES TO DECREASE MATERNAL MORTALITY

- A) *Antenatal*: Proper antenatal care with regular follow up of the mothers during pregnancy. High risk cases as diabetics and cardiac cases are seen more frequent. Correction of anaemia and malnutrition. Hospitalization of cases of pre-eclampsia. Ultrasound examination to diagnose cases of placenta praevia.
- B) *Intranatal*: (1) Delivery of all cases at hospital, if possible (normal delivery is a retrograde diagnosis); (2) blood should be available for bleeding cases; (3) Flying squad to ensure rapid transfer of urgent cases; (4) prevention of prolonged labour; (5) interference before labour becoming obstructed; (6) nothing is given by mouth during labour to avoid Mendelson syndrome if general anaesthesia is needed; (7) general anaesthesia if needed should be given by an expert anaesthetist; (8) routine use of ecbolics in the third stage of labour to reduce the incidence of postpartum haemorrhage; (9) aseptic and antiseptic measures should be followed.
- C) *Postnatal*: (1) Infected persons or carriers should not come in contact with the puerpera; (2) maintain aseptic and antiseptic measures; (3) antibiotics if indicated.

Note: Mendelson syndrome results from vomiting and inhalation of the acid gastric contents leading to severe spasm of the bronchioles and may be fatal.

CAUSES OF SUDDEN MATERNAL DEATH AFTER LABOUR

- 1) Shock due to any cause as uterine rupture or inversion;
- (2) severe haemorrhage;
- (3) amniotic fluid embolism;
- (4) postpartum eclampsia;
- (5) heart failure;
- (6) acute renal failure as in abruptio placentae;
- (7) severe infection;
- (8) anaesthetic complications as Mendelson syndrome;
- (9) other causes as coronary thrombosis and diabetic coma.

POSTNATAL CARE

Postnatal care includes the following:

- I. Immediate care after delivery.
- II. Care after transfer to postnatal ward.
- III. Postnatal visit.

IMMEDIATE CARE

The woman is kept under observation in the labour ward for at least one hour or more after delivery until the blood pressure is satisfactory, vaginal bleeding has ceased, and there are no abnormalities needing close observation. Fluids according to choice are provided.

CARE AFTER TRANSFER TO THE POSTNATAL WARD

1. Observations

- a. Vital signs. The pulse, temperature, and blood pressure are recorded every 4 hours for 24 hours then twice daily.
- b. The uterine fundus is palpated daily to check for normal involution.
- c. The vulva and perineum are checked daily to note the amount, colour, and odour of the lochia and the state of the episiotomy. After each micturition and defecation the vulva and perineum are washed with antiseptic solution from before backwards, dried, and a sterile vulval pad is applied. An infrared lamp applied to the perineum is soothing for pain and hastens healing of the episiotomy. It is used twice daily. If there is a haematoma ice packs are used.
- d. Examination of the legs daily for deep venous thrombosis (tenderness in the thigh or calf, swelling of the leg, or temperature difference).
- e. The mental state of the mother and signs of depression should be noted (postpartum blues).
- f. Observation of the fetus for jaundice and condition of the umbilical stump.

2. The haemoglobin level

It is checked next day after delivery to exclude anaemia.

3. Diet

It should be high in protein and vitamins, with adequate amount of fluids.

4. Rest

- a. Early ambulation is encouraged by allowing the woman out of bed within a few hours after delivery. For at least first ambulation patient is accompanied by the nurse. Early

ambulation reduces retention of urine, constipation, deep venous thrombosis and pulmonary embolism.

- b. Rest in bed in the semisitting position to encourage drainage of the lochia.

5. Exercises

- a. Breathing exercises to reduce the risk of deep venous thrombosis.
- b. Pelvic floor exercise is started on the third day postpartum if there is no episiotomy. The patient is asked to contract and relax the pelvic floor 3 times to be increased gradually. This is done 5 times daily. Abdominal exercises are started after one week.

6. Breasts

Should be inspected daily for cracking of nipples, inflammation or abnormal engorgement. Routine cleansing of the nipple and areola before and after each feeding using boric lotion or warm water and soap. An emollient cream may be used.

7. Bowels

Constipation is treated by increasing fluid intake, diet rich in fibre in the form of vegetables, fruits, and bran. Glycerin suppositories or a mild laxative as Agarol can be given. Haemorrhoids may be troublesome and can be relieved by suppositories, local ointment and astringent preparations as magnesium sulphate.

8. Bladder

Patient is encouraged to micturate frequently. Retention of urine may occur due to urethral bruising, reflex inhibition from a painful perineal or abdominal wall, or temporary paralysis following epidural analgesia. A catheter is passed under aseptic conditions. If residual urine is high (more than 100 ml) the catheter should be left for 24 hours. All women catheterized during or after labour should have a mid-stream urine specimen 2 days later for culture and sensitivity test.

9. Pain relief

Following delivery of the placenta the uterine muscle contracts, and the woman experiences cramps known as afterpains. These may continue for 3 or 4 days. The after pains are more severe in the multipara. Also mothers who breast-feed their infants are likely to have more afterpains which are caused by release of oxytocin as a result of suckling. An analgesic is given.

10. Immunization

- a. The nonimmunized rhesus negative mother who delivered a rhesus positive infant should receive anti-D immune globulin. The dose is 300 micrograms given IM within

72 hours after delivery. However, a Kleihauer test is done 24 hours after the injection to be sure that the dose given has destroyed all fetal red cells in the maternal circulation.

- b. If the mother is Hepatitis B negative, the infant receives active immunization with Hepatitis B vaccine. If the mother is a carrier, the infant receives both active immunization and passive immunization with Hepatitis B immune globulin (0.5–1 ml IM).
- c. Women who are not already immune to rubella are vaccinated before discharge. Pregnancy should be avoided for 3 months as it is a live attenuated vaccine.

11. Education

- a. Instructions are given to the mother regarding her own self-care and care of the baby.
- b. Advantages of breast-feeding are explained to the parents.
- c. During postpartum hospital stay, the method of contraception is discussed with the patient.
- d. If perineum is intact, coitus can be started 3 weeks after delivery, based upon patient desire and comfort.
- e. Mother is informed that if she is not breast-feeding menstruation usually occurs within 6–8 weeks after delivery.
- f. Patient is instructed to lie on her abdomen for one hour daily for 2 weeks starting 2 weeks after delivery to prevent puerperal retroversion.

12. Visitors

Only the husband and close relatives are allowed during the first 72 hours. Infected persons or carriers should not come in contact with the puerpera.

13. Discharge

In uncomplicated vaginal delivery, the patient is usually discharged after 48 hours.

III. POSTNATAL VISIT

Some obstetricians examine the patient during the 3rd postpartum week, while others give appointment 4–6 weeks after delivery. Early examination may detect problems and provides moral support for the mother. Postnatal examination includes:

1. History

The patient is asked about:

- a. Vaginal bleeding or discharge.
- b. Breast disorders.
- c. Urinary symptoms.
- d. Gastrointestinal symptoms.

- e. Backache. It is a problem in at least 10% of cases and is usually due to strain of sacroiliac joint.

2. General Examination

To exclude hypertension and breast abnormalities.

3. Abdominal Examination

For lax abdominal wall, subinvolution of uterus, and caesarean section scar if present.

4. Pelvic Examination

- a. Inspection of the vulva and perineum for the episiotomy wound, vaginal bleeding, discharge, gaping of the introitus, prolapse, stress incontinence, and tone of levator ani muscles.
- b. Vagina. For vaginitis.
- c. Cervix. For erosion, cervicitis, and lacerations. A cervical smear is taken to exclude cervical intraepithelial neoplasia if the last smear was taken one year or more before.
- d. Uterus. For size, position, and any abnormality.
- e. Adnexa. For any swelling.

5. Instructions

- a. The method of contraception to be practiced.
- b. Patient is informed that temporary dyspareunia may occur due to a tender episiotomy wound, and dry vagina. Dryness of the vagina occurs in a breast-feeding mother as prolactin inhibits the ovaries and leads to diminished oestrogen production. There may be decreased sexual response due to transfer of emotional interest to the infant.

6. Management

- a. Uterine prolapse. If detected during the puerperium, a ring pessary is applied for 3 months until the supporting cervical ligaments involute and restore their tone.
- b. Puerperal retroversion. The uterus is corrected at first then a Hodge or Smith pessary is introduced to keep it anteverted and is left for 3 months.
- c. Cervical erosion (ectopy). Postpartum erosion is left for 3 months as spontaneous cure may occur.
- d. Subinvolution of uterus. See puerperium

Note: Postnatal blues. The condition occurs in more than 50% of women after delivery. It starts 2 to 4 days after delivery and lasts for 2 to 7 days. It is a mood disorder, and the symptoms include headache, sleep disturbances, poor concentration and attacks of crying. It is a reaction to the physical and mental stress of labour, discomforts of the early puerperium, lack of sleep, anxiety about caring for the baby, and alteration in hormone levels (withdrawal of oestrogen and progesterone after delivery of the placenta). This mild disorder is self-limited. Treatment by reassurance and oestrogen skin patches.

THE NEWBORN

FETAL BIRTH INJURIES

I. CEPHALHAEMATOMA

It is a subperiosteal haematoma over a cranial bone. It occurs after birth trauma as forceps or ventouse delivery but may occur after a normal and easy delivery. Normally the blood is absorbed gradually but it may become organized or even ossified forming bone. An X-ray film of the skull should be obtained to exclude fracture; as well as study of coagulation factors to exclude blood disease as thrombocytopenia.

<i>Cephalhaematoma</i>	<i>Caput Succedaneum</i>
1. It is present at birth but it may not appear except 1 or 2 days after delivery due to slow accumulation of blood. It disappears within 2 weeks but it may take a longer time depending upon its size. 2. It is localized and limited to the borders of the affected bone. 3. The skin over it is normal. 4. There is no pitting on pressure. 5. Skull fracture may be present.	1. It is present at birth and disappears within 1 or 2 days after delivery. 2. It is diffuse and overlies more than one bone. 3. The skin may be dark red and ecchymotic. 4. There is pitting on pressure. 5. No skull fracture.

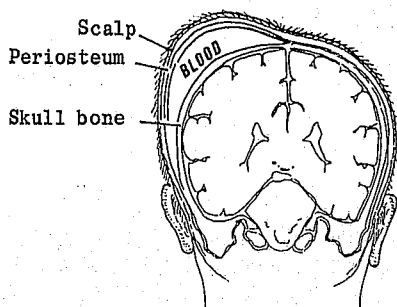


Fig. 116. Cephalhaematoma

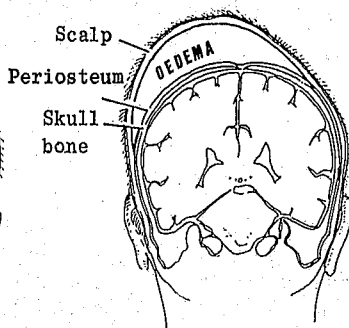


Fig. 117. Caput Succedaneum

Treatment

Wait for spontaneous absorption. Vitamin K₁ (1 mg) is given I.M. If it does not absorb within two weeks it is aspirated to avoid chronicity.

II. INTRACRANIAL HAEMORRHAGE

The causes are (1) marked compression of the head by forceps or contracted pelvis; (2) rapid compression due to rapid delivery of the aftercoming head in breech and precipitate labour; (3) blood diseases as haemophilia and thrombocytopenia; (4) prematurity, due to fragility of the blood vessels, soft cranial bones and physiological hypoprothrombinaemia; (5) asphyxia leading to rupture of blood capillaries; (6) skull fracture.

Site of Bleeding

The bleeding is usually due to rupture of the great cerebral vein of Galen or the straight sinus; due to a tear in the dura at the junction of the falx cerebri with tentorium cerebelli. The haemorrhage may be extradural, subdural, subarachnoid, periventricular, intraventricular or in the brain substance.

Clinical Picture

1. Stillbirth; or
2. The infant is drowsy, irritable, refuses suckling and produces sudden sharp cries. Convulsions, rigidity and paralysis may occur. The breathing is irregular with attacks of apnoea and cyanosis. Bulging of the anterior fontanelle is characteristic. Ultrasound and CT scan can locate the site of haemorrhage.

Prophylaxis

1. Vitamin K₁ is given routinely to all infants immediately after delivery to guard against intracranial haemorrhage.
2. Dexamethasone is given when labour starts before 34 weeks to reduce the incidence of respiratory distress syndrome and periventricular haemorrhage.

Treatment

1. Vitamin K₁ (1 mg I.M.).
2. Oxygen for cyanosis.
3. Sedatives for irritability.
4. Luminal for convulsions.
5. Hypertonic solutions per rectum, as 10% sodium chloride to diminish cerebral oedema.
6. Antibiotics to guard against pulmonary infection.
7. Lumbar puncture may be done if the anterior fontanelle is bulging to reduce the high intracranial tension.

8. No oral feeding for 72 hours.

III. NERVE INJURIES

1. Facial Palsy

Malapplication of forceps leads to direct pressure on the nerve at the stylomastoid foramen causing oedema and haemorrhage around the facial nerve fibres.

2. Brachial Palsy

It results from traction on the head or excessive lateral flexion of the neck during delivery of the shoulders. In Erb's palsy the roots of cervical nerves 5 and 6 are damaged. The upper limb hangs beside the body, the forearm is extended and pronated and the hand flexed (waiter tip position). In Klumpke palsy the roots of cervical nerves 7 and 8 and thoracic 1 are damaged with flaccid paralysis of the hand.

IV. SKELETAL, MUSCLE AND VISCERAL INJURIES

These usually complicate breech delivery.

FETAL ASPHYXIA

I. INTRAUTERINE (ANTENATAL) ASPHYXIA

Aetiology

A) Maternal Causes

Maternal hypoxia from any cause; anaemic hypoxia due to severe anaemia, or excessive haemorrhage, hypoxic hypoxia as in pulmonary diseases, stagnant hypoxia as in heart failure and shock, and histotoxic hypoxia as in cyanide and barbiturate poisoning.

B) Placenta Causes

1. Compression of the placenta as in prolonged or obstructed labour.
2. Premature separation of the placenta as in case of placenta praevia or abruptio placentae.
3. Placental insufficiency as in case of pre-eclampsia, chronic hypertension, chronic renal disease, diabetes mellitus and postterm pregnancy.

C) Causes in the Umbilical Cord

1. A true knot, or tight coiling of the cord around the fetus.
2. Compression of prolapsed cord.
3. Compression of the cord by the forceps.
4. Rupture of vasa praevia.

D) Fetal Causes

Prolonged compression or excessive moulding of the fetal head during labour. This interferes with the fetal cerebral circulation leading to hypoxia of the respiratory centers. This compression may be caused by contracted pelvis, forceps or rigid perineum.

Clinical Picture

A) During Pregnancy

Fetal asphyxia during pregnancy leads to intrauterine growth restriction, congenital malformation, fetal distress and fetal death.

B) During Labour

Fetal asphyxia leads to fetal distress and fetal death. Fetal distress is diagnosed by:

1. Changes in fetal heart rate (FHR).
2. Presence of meconium in amniotic fluid in cephalic presentation due to increased intestinal movements and relaxation of external anal sphincter caused by hypoxia. However, this is not a reliable sign as a mature fetus can pass meconium in absence of fetal distress also the hypoxic preterm baby may not pass meconium.
3. Fetal acidosis detected by taking blood samples from fetal scalp during labour. A pH below 7.2 indicates fetal acidosis due to hypoxia.
4. Excessive fetal movements. This is a terminal sign.

Changes in FHR

- a) *Intermittent auscultation*: Using the fetoscope or Doptone (Sonicaid). The signs of fetal distress are; (1) tachycardia with acceleration of FHR above 160 beats per minute; (2) bradycardia with slowing of FHR below 120 bpm; (3) irregularity in FHR. Auscultation should be done immediately after the end of a contraction.
- b) *Continuous electronic monitoring of FHR*: The signs fetal distress are; (1) tachycardia above 160 bpm; (2) bradycardia below 120 bpm; (3) late decelerations (type 2 dips); (4) absence of beat to beat variation; (5) prolonged and persistent variable decelerations.

Note: Mild degree of hypoxia causes acceleration while severe degree causes slowing of fetal heart rate.

Treatment

If signs of fetal distress occur during labour the fetus is delivered immediately either vaginally or abdominally according to the condition. Oxygen is given to the mother while preparing for delivery.

Note: Meconium consists of (a) the secretions of the fetal gastrointestinal tract including bile; (b) sloughed intestinal epithelial cells; and (c) swallowed amniotic fluid containing debris as fetal hair, vernix caseosa and fetal skin cells. Meconium may be thin or thick. Thin meconium is not harmful to the newborn. Thick meconium is harmful and can lead to meconium aspiration syndrome which is characterized by (a) obstruction of the air passages and alveoli by meconium; (b) chemical pneumonitis; and (c) persistent pulmonary hypertension of the newborn. The presence of meconium in amniotic fluid is explained by (a) fetal hypoxia; (b) a mature fetus can pass meconium in absence of hypoxia; and (c) transient compression of umbilical cord leading to vagal stimulation.

II. POSTNATAL ASPHYXIA (ASPHYXIA NEONATORUM)

This means failure of the fetus to start normal respiration after delivery.

Aetiology

1. Persistence of intrauterine asphyxia after delivery.
2. Faults in the respiratory centers due to:
 - a) Poor development in the preterm baby.
 - b) Depression by anaesthesia and drugs as pethidine.
 - c) Damage by cerebral haemorrhage or oedema.
3. Obstruction of the air passages caused by:
 - a) Amniotic fluid, blood, mucus or meconium.
 - b) Congenital atresia of larynx or trachea.
 - c) Neck tumours.
4. Abnormalities of the lungs:
 - a) Congenital atelectasis.
 - b) Lack of lung surfactant leading to respiratory distress syndrome.
5. Weakness of respiratory muscles due to congenital debility or prematurity so the muscles are unable to start respiration.

Diagnosis

The Apgar Score: One minute after delivery of the infant, the following 5 signs are evaluated and each given a score of 0, 1 or 2. Apgar score of 7–10 is normal. A score of 6 or less indicates fetal asphyxia which needs resuscitation. Apgar 0–3 indicates severe asphyxia and 4–6 indicates moderate and mild asphyxia. The score is determined again 5 minutes after delivery. A low score at 5 minutes at birth indicates increased risk of infant mortality and morbidity.

Sign	0	1	2
A. Appearance (colour)	Pale or Blue	Body pink; limbs blue	Completely pink
P. Pulse (heart rate)	Absent	Below 100	Over 100
G. Grimace (response to stimulus as catheter in nostril)	No response	Grimace	Sneeze or cough
A. Activity (muscle tone)	Flaccid	Some flexion of limbs	Active movement
R. Respiration	Absent	Slow or irregular	Regular or crying

Management

Resuscitation of the newborn is carried out as follows:

1. Airway suctioning: Immediately after delivery of the baby the nose, mouth and pharynx are suctioned by a suction catheter.
2. Breathing: If respiration does not start one minute after delivery oxygen is given under pressure using a bag and a mask applied over the face. The rate of bagging is 40–60/minute. If this does not stimulate respiration after 30 seconds an endotracheal tube is passed using fetal laryngoscope. Any mucus, blood or meconium is removed by suction and oxygen is allowed to flow under intermittent positive pressure to expand the alveoli.
3. Cardiac resuscitation: If the heart rate is less than 60/minute do external cardiac massage. Two fingers are applied over midsternum to press at a rate of 120 times/minute and epinephrine may be injected directly into the heart.
4. Drugs: Naloxone (10 micrograms/kg body weight) is injected into the umbilical vein or IM or subcutaneously if the mother was given pethidine within 2 hours of delivery. Dose is repeated every 2–3 minutes if necessary. When recovery is delayed, sodium bicarbonate (1 mEq/Kg) is given into the umbilical vein or peripheral vein to treat acidosis. Antibiotics are given when recovery has been difficult to guard against infection.
5. The baby is kept warm to prevent cold stress (cooling increases the metabolic rate and oxygen requirement, increases the apnoeic attacks, causes hypoxia, hypoglycaemia and acidosis).
6. Efforts at resuscitation are continued as long as the heart is beating.

THE RESPIRATORY DISTRESS SYNDROME (RDS) (HYALINE MEMBRANE DISEASE)

AETIOLOGY

The RDS occurs in preterm babies, infants of diabetic mothers and infants delivered by caesarean section. It is due to absence or presence of a small amount of the lung surfactant. The surfactant is formed by alveolar cells (type II pneumocytes). It reduces the surface tension or stickiness of alveolar walls and helps expansion of the alveoli during inspiration and prevents their collapse during expiration. The surfactant is a group of phospholipids which include lecithin, sphingomyelin, phosphatidylglycerol and other phospholipids. If the lecithin/sphingomyelin ratio in the amniotic fluid is 2:1 or more the RDS is not liable to occur in the newborn. The figure should be 3.5:1 if the mother is diabetic. Also the appearance of phosphatidylglycerol (more than 2 mg%) is more reliable in detecting lung maturity especially in diabetes mellitus as it is the last lung surfactant to develop and is not affected by the presence of blood or meconium.

PATHOLOGY

Microscopically the lungs show areas of atelectasis. The alveoli are collapsed and the alveolar ducts are overdistended. The alveoli, alveolar ducts, and respiratory bronchioles are lined by a hyaline membrane which is a homogeneous pink eosinophilic membrane composed mainly of fibrin, and resembles clotted plasma. The pulmonary capillaries are congested. It is believed that hypoxia resulting from RDS causes exudation of fibrin from the pulmonary capillaries.

CLINICAL PICTURE

Asphyxia generally appears soon after birth and becomes progressively more severe. Respiration becomes rapid, difficult and grunting with retraction of the suprasternal, intercostal and subcostal areas during inspiration. Attacks of apnoea and cyanosis occur. X-ray examination reveals diffuse granular opacities throughout both lungs (areas of atelectasis). Sometimes the bronchial tree is clearly seen because of its content of air. The syndrome may cause death within a few hours or days.

TREATMENT

1) The infant is nursed in an incubator with high humidity. Oxygen is given for hypoxia and cyanosis; (2) antibiotics to prevent pulmonary infection; (3) correction of acidosis by intravenous sodium bicarbonate and glucose through the umbilical vein; (4) feeding by the intravenous or intragastric drip of 10% glucose solution until recovery starts; (5) treatment with human surfactant (extracted from amniotic fluid), or animal

surfactant (extracted from bovine, cow, or calf lung) or synthetic surfactant composed of phospholipids. The surfactant is instilled endotracheally. It is given prophylactically or after development of symptoms.

Notes:

- At 34 weeks of gestation the L:S ratio is 2 or more, and the incidence of RDS is low (only 2-7%).
- The L:S ratio is more reliable than measuring lecithin alone, because the ratio is not affected by increased or decreased volume of amniotic fluid. The L:S ratio is not reliable in the presence of blood or meconium as these contain lecithin and change the ratio.
- Phosphatidylglycerol starts to appear at 35 weeks of pregnancy.
- Corticosteroids, thyroxine, and prolactin increase the production of lung surfactant and accelerate lung maturity. Insulin reduces its production and delays lung maturity.
- Corticosteroids (dexamethasone or betamethasone) start their action one day (24 hours) after the injection of the drug and action continues for 7 days.
- If corticosteroids are given after 34 weeks of pregnancy, the incidence of RDS is not reduced, so they are given between 24 and before 34 weeks.

STILLBIRTH

This means that the fetus is born dead after 20 weeks of pregnancy or if the fetal weight is 500 g or more. The causes are:

1. Antenatal or intrauterine fetal death. This means death of the fetus during pregnancy before the onset of labour.
2. Intranatal death or death of the fetus during labour.

1. INTRAUTERINE FETAL DEATH (MACERATED STILLBIRTH)

Aetiology

A) Maternal Causes

1. Pregnancy induced hypertension.
2. Chronic hypertension.
3. Chronic renal disease.
4. Diabetes mellitus.
5. Maternal hypoxia due to any cause as anaemia and congenital heart disease.
6. Severe malnutrition.
7. Autoimmune diseases as systemic lupus erythematosus.
8. Antiphospholipid antibody syndrome. The presence of antiphospholipid antibodies as anticardiolipin antibodies or lupus anticoagulant. These immunoglobulins may lead to placental thrombosis and fetal death.
9. Smoking leading to fetal asphyxia and even death.
10. Drugs as folic acid antagonists and cytotoxic drugs.

B) Placental Causes

1. Small placenta.
2. Placental thrombosis and infarction.
3. Separation of the placenta in placenta praevia and abruptio placentae.
4. Circumvallate placenta.
5. True knot of the umbilical cord and loops of cord around fetal neck or body (fetal hypoxia).
6. Rupture of vasa praevia.
7. Marginal and velamentous insertion of the cord.
8. Single umbilical artery.
9. Chorioamnionitis leading to fetal infection. This may follow premature rupture of membranes.

C) Fetal Causes

1. Chromosomal abnormalities as trisomies.
2. Congenital fetal malformation as anencephaly.
3. Fetal infections which may be bacterial (as listeriosis and tuberculosis), viral (as rubella and cytomegalovirus), protozoal (as toxoplasmosis and malaria) and spirochetal in case of syphilis.
4. Multiple pregnancy. Twin-to-twin transfusion syndrome and due to placental insufficiency as the placental mass is small in relation to the fetal mass.
5. Postterm pregnancy which leads to placental insufficiency.
6. Erythroblastosis fetalis (destruction of fetal red cells leads to anaemia and heart failure).
7. Feto-maternal haemorrhage leading to fetal exsanguination. May follow trauma but mostly due to unknown cause.

D) Idiopathic

In the majority of cases (30–50%) no cause is found.

Diagnosis

Symptoms

1. Gradual disappearance of pregnancy symptoms as nausea, vomiting, and breast symptoms.
2. Milk secretion may start spontaneously from the breast (Page 43).
3. Failure of the abdomen to increase in size.
4. Failure to feel fetal movements or cessation of fetal movement, if previously present.
5. A dark brown vaginal discharge may occur (prune juice discharge).

Signs

1. The uterus is smaller than the period of amenorrhoea and fails to enlarge on repeated examination. However, about 4 weeks must pass before changes in uterine size can be reliably detected which is not practical.
2. The fetal parts are difficult to feel because of loss of muscle tone and fetal heart sounds are not heard.

Investigations

1. Pregnancy Test

It becomes negative within 2 weeks after fetal death but may remain positive for a longer time if there is still living chorionic tissue.

2. Ultrasonic Examination

Absence of cardiac activity, absence of fetal movements, overlapping of skull bones, scalp oedema and evidence of fetal maceration.

3. X-ray Examination.

This may show:

- a) *Spalding sign*: The bones of the skull overlap each other due to softening of the brain and absorption of the cerebrospinal fluid. The sign becomes positive one week after death. A similar picture is seen during labour due to moulding.
- b) *Ball sign*: The fetus is rolled up like a ball due to loss of muscle tone.
- c) *Halo sign* due to scalp oedema.
- d) Angulation of the spine due to loss of tone of paraspinal muscles.
- e) Collapse of the thorax.
- f) Rarefaction of bones giving a faint shadow.
- g) Nitrogen gas appears in fetal heart or large blood vessels (*Robert sign*). It is the earliest sign and can appear within 48 hours of death (caused by blood degeneration).
- h) X-ray may reveal the cause of death as syphilitic affection of bones.

Complications

1. Intrauterine infection.
2. Disseminated intravascular coagulation and hypofibrinogenaemia.

Treatment

We wait 4 weeks after fetal death so that the uterus may expel its contents spontaneously. If this does not occur we try medical induction by oxytocin drip or by prostaglandins. If these measures fail we can try intra-amniotic injection of hypertonic 20%

sodium chloride solution or 40% urea solution in 5% glucose, or the uterus is evacuated abdominally. Immediate interference is indicated if there is infection, bleeding, or if the patient is anxious. Blood is tested for its fibrinogen content before induction.

Notes:

- Maceration means peeling of the skin and it starts 12 hours after fetal death.
- Methods of administration of prostaglandins: see missed abortion (page 44).
- To diagnose feto-maternal haemorrhage we do the Kleihauer test which detects fetal red cells in maternal blood. It is done in all cases of IUFD particularly when no cause is apparent.

II. INTRANATAL DEATH (FRESH STILLBIRTH)

It is death of the fetus during labour.

Aetiology

1. Intrauterine asphyxia.
2. Intracranial haemorrhage.
3. Congenital pneumonia due to premature rupture of membranes or prolonged labour.
4. Birth trauma as fracture of the cervical spine, rupture of liver or spleen.

NEONATAL DEATH

It is death of the infant during the first 4 weeks after delivery. Early neonatal death is death of the infant during the first 7 days after birth. Late neonatal death is death of the infant after 7 days, but before 29 days.

AETIOLOGY

- 1) Prematurity, the main cause (50%); (2) asphyxia neonatorum; (3) birth injuries; (4) congenital abnormalities; (5) infections as syphilis or infection of the umbilical stump; (6) haemolytic diseases and erythroblastosis fetalis; (7) respiratory distress syndrome.

PERINATAL MORTALITY RATE

It is the number of stillbirths plus the neonatal deaths per 1000 total births. It is 8-12 per 1000 in developed countries.

PRETERM LABOUR (PREMATURITY)

DEFINITION

Preterm labour means labour occurring after viability of the fetus (20 weeks) and before 38 weeks of pregnancy (37 completed weeks).

Note:

- *Preterm infant:* An infant born before 38 weeks of pregnancy. In the past, the term premature infant was used.
- *Low birth weight:* Fetal weight at birth is less than 2500 g. This low birth weight is due to either preterm labour or intrauterine growth restriction (small-for-dates).
- *Very low birth weight:* Fetal weight at birth is less than 1500 g.
- *Extremely low birth weight:* Fetal weight at birth is less than 1000 g.

INCIDENCE OF PRETERM LABOUR

5–10% of all deliveries.

AETIOLOGY OF PRETERM LABOUR**A) Maternal Causes**

1) Pregnancy induced hyperextension; (2) chronic hypertension; (3) chronic renal disease; (4) diabetes mellitus; (5) maternal hypoxia due to any cause as anaemia and congenital heart disease; (6) malnutrition; (7) smoking; (8) asymptomatic bacteriuria and urinary tract infection; (9) psychogenic disturbances; (10) uterine abnormalities as hypoplasia and incompetent cervix; (11) pelvic tumour as fibroid or ovarian cyst; (12) polyhydramnios; (13) premature rupture of membranes; (14) previous preterm labour; (15) threatened abortion increases the risk of preterm labour in that pregnancy; (16) abdominal surgery in the second half of pregnancy; (17) maternal age: pregnancy occurring before the age of 20 or for the first time in a woman over 35 years; (18) low socioeconomic class; (19) low pre-pregnancy weight <45 kg; (20) trauma; (21) black race.

B) Placental Causes

1) small placenta; (2) placental thrombosis and infarction; (3) separation of the placenta in placenta praevia and abruptio placentae; (4) circumvallate placenta; (5) true knot of the cord or loops of cord around fetal neck or body (fetal hypoxia); (6) rupture of vasa praevia; (7) chorioamnionitis which stimulates uterine contractions.

C) Fetal Causes

1) Chromosomal abnormalities; (2) congenital fetal malformation; (3) multiple pregnancy; (4) erythroblastosis fetalis; (5) congenital adrenal hyperplasia (excess cortisol).

D) Induction of Preterm Labour

In complicated pregnancy as intrauterine growth restriction and diabetes mellitus.

E) Idiopathic

In the majority of cases (30–50%), no cause is found.

Note: Infection of the fetal membranes, i.e., chorioamnionitis leads to preterm labour as the micro-organisms produce phospholipase A2 enzyme which leads to formation of prostaglandins.

Also, bacterial endotoxins stimulate decidual cells to produce cytokines and these lead to production of prostaglandins which stimulate uterine contractions. In addition infection causes weakness and rupture of the membranes due to production of proteases (proteolytic enzymes) and the inflammatory reaction. Rupture of membranes stimulates uterine contractions.

CLINICAL PICTURE

The main features of the preterm baby are:

1. Weight may be less than 2.5 kg (mature baby, 3.2 kg).
2. Length is diminished: less than 45 cm (mature, 50 cm).
3. Vitality is diminished, infant is drowsy and the cry is weak.
4. Suckling is weak or absent.
5. Hypothermia, because of increased heat loss due to lack of development of the heat regulating center, lack of subcutaneous fat, diminished muscular activity and large surface area in relation to body weight.
6. Respiration is irregular, with attacks of apnoea and cyanosis.
7. Skin is thin, red and wrinkled due to lack of subcutaneous fat.
8. Little or no vernix caseosa. It is a fatty layer produced by the sebaceous glands of fetal skin to prevent its maceration by amniotic fluid.
9. If born before 32 weeks lanugo hair will be present all over the body.
10. Finger nails are soft and short and do not reach the tips of fingers.
11. Testes are undescended.

COMPLICATIONS OF PRETERM BABY

1. Haemorrhage as intracranial haemorrhage (low prothrombin level due to immaturity of liver, soft cranial bones and fragile blood vessels).
2. Pulmonary complications: (a) Respiratory distress syndrome due to lack of lung surfactant. The most common complication of prematurity; (b) aspiration of amniotic fluid or meconium (meconium aspiration syndrome); (c) pneumonia. Infection is common in the preterm baby due to deficiency of maternal antibodies and poor antibody formation.
3. Gastrointestinal complications: Gastroenteritis and necrotizing enterocolitis.
4. Blood disorders: (a) Hypoprothrombinaemia (b) anaemia due to poor store of iron in the fetal liver. Iron is normally stored in the last 3 months of pregnancy. Also the red blood cells are easily destroyed (haemolytic anaemia); (c) hyperbilirubinaemia, jaundice and kernicterus due to immaturity of the liver and excessive destruction of red cells.

5. Metabolic disorders: (a) Malnutrition due to poor suckling, weak power of absorption and poor stores of minerals and vitamins; (b) hypocalcaemia and rickets; (c) hypoglycaemia due to lack of store of glycogen in the liver; (d) hypothermia.
6. Mental retardation.
7. Retrolental fibroplasia and blindness may complicate high oxygen therapy.
8. There is increased incidence of congenital malformations among the preterm babies (fetal anomalies lead to preterm labour).

MANAGEMENT

A) Prophylactic Treatment

Preterm labour is prevented by:

1. Proper antenatal care with treatment of anaemia, malnutrition, asymptomatic bacteriuria, urinary tract infection and other conditions which predispose to preterm labour. Smoking is stopped.
2. Cervical cerclage for cervical incompetence.
3. Women with previous preterm labour (high risk) are screened and treated for bacterial vaginosis.
4. More periods of rest for those who are liable to have preterm labour as cases of multiple pregnancy and polyhydramnios.
5. Selective embryo reduction in multifetal pregnancy (see multiple pregnancy).
6. Serial measurements of fetal fibronectin in the cervicovaginal secretions every 2 weeks from 24 to 34 weeks in women at high risk of preterm labour. If the level is above 50 nanograms/ml preterm labour will occur in 30% of cases within one week, and prophylactic measures are taken. If the test is negative preterm labour occurs in less than 1% of cases during the next week. Normally fetal fibronectin is present in cervicovaginal secretions before 20 weeks of pregnancy and at term. It is believed that it causes adhesion of placenta and membranes to the decidua.

B) Treatment of Premature Uterine Contractions by:

1. Rest in bed. It increases uteroplacental blood flow and this decreases uterine activity. Rest in bed is the "Chicken soup" of obstetrics and recommended for most pregnancy problems.
2. Sedatives as diazepam (Valium).
3. Hydration of the patient. Normal saline or 5% glucose solution is given I.V. (500 ml given in 20 minutes). Water decreases the secretion of antidiuretic hormone and oxytocin from the posterior pituitary gland. It also increases uteroplacental blood flow and this diminishes the uterine activity.

4. A tocolytic drug is given if pregnancy is less than 34 weeks (a beta-mimetic drug as ritodrine or magnesium sulphate solution are commonly used). The drug is given for 48 hours, i.e., short-term tocolysis, to delay labour until dexamethasone stimulates lung maturity.
5. Corticosteroids. If pregnancy is less than 34 weeks to stimulate maturation of the lungs (dexamethasone 12 mg I.M. every 12 hours for 2 doses and can be repeated weekly).

C) During Labour

1. Delivery must be at hospital.
2. To prevent group B streptococcal infection in the neonate, the American College of Obstetricians and Gynaecologists recommends ampicillin 2 g IV every 6 hours until delivery for women in labour before 38 weeks.
3. Strong sedatives are avoided to avoid depression of the fetal respiratory center. Epidural analgesia can be used.
4. Episiotomy and low forceps if there is any perineal rigidity.
5. Vitamin K₁ is given to the fetus immediately after delivery; 1 mg. I.M. Then the vitamin is given orally 1 mg at the end of the first and third weeks.
6. Caesarean section if the fetus is premature breech and weighs 1000–1500 grams (28–32 weeks). This is safer than vaginal delivery.

C) After Delivery

1. The nose, mouth and pharynx should be cleared immediately by a suction catheter.
2. No bath is given so as not to remove the vernix caseosa.
3. The incubator is essential if the weight is less than 2 kg. The fetal temperature is kept at 36° C for minimal oxygen consumption. This is obtained by adjusting the temperature of the incubator between 32° C and 35° C. The smaller the infant, the higher the temperature. The humidity should be at least 75%, oxygen concentration should not exceed 35% to avoid retrolental fibroplasia.
4. Feeding: It is started early within a few hours (3 hours) after birth to avoid hypoglycaemia. The first feeds are 5% glucose solution. Mother's milk is the best food. If the infant cannot suckle it is fed by a spoon or a dropper. If it can not swallow a polythene catheter is introduced into the stomach for feeding.
5. Multiple vitamins (A, B, C, D) and iron are given after the first week.

Notes:

- Fetal fibronectin is a glycoprotein formed in the decidua and amniotic membrane and is normally present in the amniotic fluid. Normally it is not detected in the cervical or vaginal secretions after 20 weeks of pregnancy, but it is detected at term. Its presence in a cervicovaginal sample indicates rupture or separation of the membranes from the uterine wall (decidua) and in this case preterm labour may occur. A sterile dactron swab is used to scrape the

cervix outside the external os, then it is inserted into the posterior vaginal fornix to take a vaginal sample as well. The cervicovaginal specimen is examined by the ELISA test (enzyme-linked immunoabsorbent assay test).

- The benefits of dexamethasone administration in preterm labour are reduction in the incidence of RDS (by about 60%), periventricular haemorrhage, and necrotizing enterocolitis. Dexamethasone reduces the last 2 complications by preventing hypoxia which occurs secondary to RDS, and by direct effect on the periventricular and gastrointestinal blood capillaries.

PREMATURE RUPTURE OF MEMBRANES (PROM)

It means rupture of fetal membranes (amniorrhexis), before onset of labour, regardless of gestational age.

INCIDENCE

About 5-10% of all deliveries.

AETIOLOGY

1. Idiopathic. The cause is unknown in most cases.
2. Inherent weakness of the membranes due to reduction in the collagen content of the amniotic membrane.
3. Incompetent cervix.
4. Increased intrauterine pressure as in polyhydramnios and multiple pregnancy.
5. Infection of the membranes (chorioamnionitis) leads to their weakness and rupture due to inflammatory reaction and production of proteases which are proteolytic enzymes. The organisms responsible include gonococci, chlamydia trachomatis, group B streptococcus, trichomonas vaginalis, and organisms causing bacterial vaginosis.
6. Increased concentration of prolactin in the chorionic membrane.
7. When the presenting part is not fitting well against the lower uterine segment as in malpresentation, and contracted pelvis.
8. Antepartum haemorrhage due to placenta praevia or placental abruption.
9. Malnutrition and deficiency of vitamins and minerals as vitamin C, copper, and zinc, leading to weakness of the membranes.
10. Trauma in the form of external cephalic version and amniocentesis.
11. Sexual intercourse. Uterine contractions are stimulated by orgasm or seminal prostaglandins leading to rupture of membranes. Also collagenase-like enzymes present in seminal fluid weaken the membranes.
12. Smoking. It impairs protein metabolism and reduces levels of vitamin C, vitamin B12, and aminoacids.
13. Ehlers-Danlos syndrome. It is a genetic disorder of connective tissue with abnormal collagen.

DIAGNOSIS

I. History

The patient complains of a sudden escape of fluid from the vagina which continues to soak pads or underclothes. We have to ask about the amount, colour, and odour of the fluid, and the time when leakage started. The amniotic fluid has a particular smell, resembling that of semen.

II. Physical Examination

1. A sterile speculum is introduced to see amniotic fluid coming from the cervix or collecting in the posterior vaginal fornix. If no fluid is seen ask the patient to cough or press on the uterine fundus to force the fluid to appear from the cervix. If no fluid is seen we do the following tests:
 - a. Nitrazine paper test. We touch the vaginal wall with the nitrazine paper (yellow in colour). If its colour turns blue it means that the vaginal pH is above 6.5 and the membranes are ruptured (the pH of amniotic fluid is 7-7.5).
 - b. Fern test. A sample of fluid is taken from the posterior vaginal fornix using a sterile cotton-tipped applicator. The sample is spread on a clean glass slide and allowed to dry, then examined microscopically. If the fern test is positive, it indicates the presence of amniotic fluid.
 - c. Other tests include detection of alpha-fetoprotein, fetal fibronectin and diamine oxidase enzyme in the vaginal fluid. The latter 2 substances are produced by decidual cells and found in amniotic fluid.
2. A cervicovaginal swab should be taken from cervical canal and vaginal vault for culture (aerobic and anaerobic) and sensitivity test.
3. Vaginal fluid is tested for the presence of phosphatidylglycerol to detect fetal lung maturity.

III. Cardiotocography

It is carried out for half an hour to detect uterine contractions, and exclude fetal distress which may occur due to compression of the umbilical cord after rupture of the membranes. Compression can occur even in absence of cord prolapse.

IV. Sonography

It is done in every case. It shows oligohydramnios, gives idea about fetal age, presentation, and excludes fetal anomalies.

Note: Digital examination is contraindicated in PROM as it introduces infection.

COMPLICATIONS

A) Maternal

1. Maternal infection in the form of chorioamnionitis (5-15%), endometritis, maternal septicaemia, and septic shock. Chorioamnionitis is diagnosed if 2 or more of the following are present: (a) fever (38 °C or above), (b) maternal tachycardia (≥ 100 beats per minute), (c) fetal tachycardia (≥ 160 beats per minute), (d) leucocytosis ($\geq 15000/\text{cu.mm}$), (e) uterine tenderness, and (f) offensive or purulent vaginal discharge.
2. Placental abruption. It occurs in about 6% of cases. PROM predisposes to placental abruption, also the latter predisposes to PROM.

B) Fetal

1. Fetal distress due to compression of the cord even in absence of prolapse.
2. Fetal infection in the form of congenital pneumonia, and septicaemia. Group B streptococcus is the commonest cause of fetal infection as well as chorioamnionitis in PROM.
3. Intrauterine fetal death due to asphyxia (cord compression) and fetal infection.
4. Preterm labour and complications of prematurity including the respiratory distress syndrome (RDS) which is the commonest complication.
5. Fetal compression syndrome due to oligohydramnios. This leads to direct pressure of uterine wall on the fetus, and formation of adhesions between the amnion and fetal skin. Deformities involve the face, ears, hands, and feet, e.g. club foot as well as pulmonary hypoplasia.

MANAGEMENT

1. Pregnancy of 36 Weeks or More

Labour is induced with oxytocin infusion if labour pains do not start spontaneously within 24 hours after rupture of membranes (induction before 24 hours increases the rate of caesarean section). At this age the RDS is uncommon.

2. Pregnancy of 32 to Less Than 36 Weeks

The treatment is conservative. Antibiotics are given to guard against maternal infection and fetal infection with group B streptococcus. Also, antibiotics prolong the pregnancy as chorioamnionitis stimulates uterine contractions. A combination of ampicillin and erythromycin is given for one week (intravenously for 48 hours then orally for 5 days).

3. Pregnancy of 26 to Less Than 32 Weeks

Conservative treatment. Antibiotics are given. Dexamethasone or betamethasone is given to stimulate fetal lung maturity. If the patient is in labour give a tocolytic drug (as ritodrine) for 48 hours (short-term tocolysis) to give a chance for dexamethasone to stimulate lung maturity.

4. Pregnancy Less Than 26 Weeks

The fetal outcome is poor in spite of any treatment. The risks are explained to the patient, and labour is either induced or conservative management is given using antibiotics, dexamethasone, and tocolysis.

CONSERVATIVE MANAGEMENT

The idea is to prolong pregnancy to allow the fetus to mature enough to avoid RDS, and it includes the following:

1. Hospitalization with complete rest in bed.
2. Observation of the mother for:
 - a- Pulse and temperature twice daily.
 - b- The presence of uterine tenderness.
 - c- The colour and odour of vaginal discharge.
 - d- Leucocytic count every other day.
 - e- Measurement of serum C-reactive protein every other day. The level is raised (2 mg/dl or more) 1-2 days before the development of clinical signs of chorioamnionitis (normal level in pregnancy is less than 1 mg/dl).
3. Observation of the fetus. The nonstress test is done daily to diagnose fetal distress and onset of labour pains.
4. When pregnancy reaches 36 weeks labour is induced with oxytocin infusion. Also, labour is induced at any time if there is fetal distress or evidence of chorioamnionitis and when lung maturity is diagnosed by the presence of phosphatidylglycerol in vaginal fluid.

Notes for the postgraduate:

1. Nitrazine paper gives a false positive result in conditions which raise the vaginal pH as when the amniotic fluid is mixed with blood, alkaline urine, semen, cervical mucus, Trichomonas vaginitis and some antiseptic solutions.
2. Fern test gives a false positive result in the presence of blood. Blood and amniotic fluid contain sodium and potassium chloride crystals which will deposit to give a palm-leaf appearance.
3. The vaginal fluid can be tested for phosphatidylglycerol to detect lung maturity. Blood, meconium, semen, and vaginal secretions contain lecithin and alters the L/S ratio.
4. Some perform amniocentesis at the time of sonography and examine amniotic fluid by Gram stain, and culture (aerobic and anaerobic) to diagnose chorioamnionitis. Also, the fluid is examined for L/S ratio or the presence of phosphatidylglycerol. When the lung is mature, labour is induced.

5. Diamine oxidase enzyme is measured by using a paper strip placed in contact with vaginal wall.
6. The first sign of chorioamnionitis is fetal tachycardia.
7. Differential diagnosis of amniotic fluid leakage includes:
 - a- Urinary incontinence.
 - b- Vaginal discharge due to infection.
 - c- Heavy show.
 - d- Cervical mucus may be profuse in women with incompetent cervix.
 - e- Hydrorrhoea gravidarum. It is due to rupture of the chorionic membrane and escape of amniotic fluid collecting between it and the amniotic membrane which remains intact.
8. Scaling of the tear in the amniotic membrane with fibrin glue has been proposed.
9. Prophylactic antibiotics are given for one week only. Prolonged antibiotic therapy may lead to appearance of resistant bacterial strains and this increases maternal and fetal morbidity. The antibiotics used are those which act against group B streptococcus and the organisms of bacterial vaginosis.

AMNIOINFUSION

It means instillation of sterile normal saline or Ringer lactate solution into the amniotic sac.

INDICATIONS

A. During Pregnancy

Transabdominal amnioinfusion may be carried out in the following conditions:

1. After failure of external cephalic version to improve the chance of success when the procedure is repeated. 800 ml warm normal saline solution is instilled over 10 minutes. Version is attempted after the infusion or after 24 hours if the uterus becomes irritable following the infusion.
2. In case of oligohydramnios to allow better fetal evaluation by ultrasound.
3. To induce abortion in the second trimester or labour in case of intrauterine death (see termination of pregnancy).

B. During Labour

Transcervical amnioinfusion may be indicated in:

1. Cases with premature rupture of membranes to avoid compression of the umbilical cord and fetal asphyxia.
2. All cases with oligohydramnios due to any cause during labour to prevent cord compression.
3. Abnormal fetal heart rate during labour in the form of variable decelerations (cord compression), type I decelerations (head compression) and diminished or absent beat-to-beat variations.
4. The presence of thick meconium. Amnioinfusion dilutes the meconium and may prevent meconium aspiration syndrome.

5. The presence of chorioamnionitis during labour. To deliver the antibiotic locally to achieve a high concentration. The local antibiotic prevents infection of fetal skin as well as the gastrointestinal and respiratory tracts (amniotic fluid is ingested and inhaled by the fetus). Transcervical amnioinfusion with 1 g ampicillin in 500 ml saline.

CONTRAINDICATIONS

1. Type II decelerations. It indicates fetal hypoxia and acidosis.
2. Fetal scalp pH less than 7.2. It indicates acidosis.
3. Malpresentation as breech.
4. Multiple pregnancy.
5. Antepartum haemorrhage.
6. Fetal anomalies incompatible with life.

COMPLICATIONS

1. Excessive or rapid infusion can raise the intrauterine pressure leading to abnormal fetal heart rate. Relieved by draining amniotic fluid.
2. Uterine hypertonicity.
3. Infection leading to amnionitis.
4. Dehiscence of uterine scar.
5. Prolapse of umbilical cord.
6. Placental abruption.
7. Amniotic fluid embolism and maternal death.

TECHNIQUE OF INTRAPARTUM AMNIOINFUSION

The fluid used is normal saline or lactated Ringer solution. The fluid is at room temperature for term pregnancies and warmed to 37 °C for preterm pregnancies. A catheter is passed through the cervix into the uterine cavity. A double lumen catheter can be used to monitor uterine activity during infusion. Transcervical infusion is done by one of two methods:

1. Infuse 300 ml over one hour, followed by a continuous infusion at a rate of 180 ml/hour or;
2. Assess the amniotic fluid index (AFI) at first, then infuse 500 ml fluid over 20-30 minutes. After 4 hours reassess the AFI. If it is 5 cm or less (oligohydramnios) reinfuse 500 ml, if it is 5-10 cm (borderline oligohydramnios) reinfuse 250 ml.

Infusion is carried out by gravity by keeping the fluid at a height of 90 cm above the patient.

POSTTERM PREGNANCY (POSTMATURITY)

DEFINITION

It means prolongation of pregnancy 2 weeks or more beyond the expected date of delivery, so duration of pregnancy is 42 weeks or more, i.e., 294 days or more.

The term postmaturity is used when postterm pregnancy is associated with placental insufficiency and intrauterine growth restriction (postmaturity or dysmaturity syndrome).

INCIDENCE

5–10% of all pregnancies.

AETIOLOGY

In most cases the cause is unknown. However, postterm pregnancy may be associated with the following factors:

1. Genetic factors. The condition often runs in families and tends to recur in the same woman (risk of recurrence is about 50%).
2. Low oestrogen levels. Oestrogen increases the sensitivity of myometrium to oxytocin.
3. Placental sulfatase deficiency (X-linked trait). The enzyme is necessary for formation of oestriol.
4. Low oxytocin and prostaglandin levels.
5. Lack of cortisol as in anencephaly and adrenal gland hypoplasia. Cortisol may play a role in the onset of labour.
6. The presence of malpresentation, malposition of the head or cephalopelvic disproportion. The presenting part fails to press on the lower uterine segment which reflexly stimulates uterine contractions (reflex polarity).
7. Improved living standards, while malnutrition leads to preterm labour.
8. Seasonal variation. Pregnancy tends to be prolonged in summer than in winter months. The average difference is 3–4 days.

DIAGNOSIS

A) History

Duration of amenorrhoea and date of quickening.

B) Clinical Examination of the Mother

1. Diminution in the abdominal girth and maternal weight (Runge sign) compared with previous finding. This is due to diminution in the amount of liquor amnii.
2. The fetal parts are easily felt and the fetal head is large and hard.

3. X-ray. The presence of a large ossific center in the fetal cuboid bone more than 5×5 mm.
4. Sonography. Biparietal diameter 10.2 cm or more and oligohydramnios.

C) Evidence of Postterm Pregnancy in the Newborn

1. With normal placental function the baby will continue to grow and show the following features: large size (macrosomia), increased subcutaneous fat, the head is large and hard with small fontanelles, and long finger nails.
2. With placental dysfunction the baby becomes underweight, with little subcutaneous fat, the skin is wrinkled and meconium stained, and long finger nails. The baby is usually alert with "old-man" look.

COMPLICATIONS

A- Fetal

1. Placental insufficiency (placental aging) leads to intrauterine growth restriction (10-20%) and even intrauterine death.
2. Macrosomia. In some cases (about 30%) the fetus continues to grow after term. Macrosomia leads to obstructed labour, shoulder dystocia, and fetal birth injury.
3. The associated oligohydramnios causes cord compression and fetal asphyxia during labour.
4. Meconium aspiration syndrome. Fetal asphyxia causes relaxation of anal sphincter and passage of meconium in the amniotic fluid and the reduced amount of the amniotic fluid cannot dilute the meconium. Thick meconium causes obstruction of the airway.

The perinatal mortality is doubled when delivery occurs after 42 weeks, tripled after 43 weeks, and quadrupled after 44 weeks.

B- Maternal

1. Uterine atony is common leading to prolonged labour and postpartum haemorrhage.
2. Obstructed labour and injury to maternal tissues because of macrosomia.
3. Increased rate of caesarean section (double the normal rate) because of fetal asphyxia, and obstructed labour and if induction of labour is carried out on unripe cervix.

MANAGEMENT

We interfere at 42 weeks as follows:

I. If the cervix is favourable (ripe)

Labour is induced by rupture of membranes and oxytocin infusion. Some obstetricians interfere at 41 weeks.

II. If the cervix is unfavourable (unripe)

Induction of labour in this case by the above method increases the rate of caesarean section, so we have one of two options:

1. To use prostaglandin vaginal tablet or gel to ripen the cervix and induce labour or;
2. We carry out expectant management. In this case we perform the modified biophysical profile twice weekly and vaginal examination once weekly. The modified biophysical profile includes the nonstress test (NST) and amniotic fluid index (AFI). Labour is induced if (a) the cervix becomes ripe, (b) the NST is nonreactive, (c) the AFI is 5 cm or less (oligohydramnios), (d) fetal weight becomes 4 kg.

Note: Favourable or ripe cervix means the cervix is short, soft, and partially dilated.

INTRAPARTUM MANAGEMENT

1. Continuous intrapartum fetal monitoring.
2. Observe for the presence of thick meconium.
3. Amnioinfusion therapy is considered if there is thick meconium or oligohydramnios.
4. Fetal nose, mouth, and pharynx are aspirated after delivery of fetal head and before delivery of its body. Aspiration of the trachea using endotracheal tube is carried out after delivery of the infant to guard against meconium aspiration syndrome.

Notes:

- 25-50% of cases diagnosed as postterm pregnancies are not true postterm due to inaccurate menstrual data due to poor memory, irregular cycles, lactational amenorrhoea or contraceptive pills taken before pregnancy (may cause delayed ovulation).
- Only 10% of pregnancies at 42 weeks have a ripe cervix.
- Meconium is found in 25-30% of postterm pregnancies, i.e., four times the incidence at term.

ASSESSMENT OF THE FETAL STATE IN PREGNANCY

I. INVESTIGATIONS TO DIAGNOSE CONGENITAL FETAL ABNORMALITIES (PRENATAL DIAGNOSIS)

1. Chorionic Villus Biopsy (CVB)

It is performed transcervical or transabdominal between 8 and 12 weeks of pregnancy. It is done without anaesthesia under ultrasound control. A sterile plastic or metal catheter is directed towards the placental site to aspirate chorionic villi (10-12 mg). Sometimes a biopsy forceps is used. The chorionic (fetal) cells are cultured and examined for: (a) chromosomal abnormalities as in Down syndrome; (b) examination of DNA which makes up the chromosomes, helps to diagnose genetic abnormalities as thalassemia and sickle-cell disease; (c) to study absent enzymes to diagnose metabolic disorders as Gaucher disease. As the chorionic cells divide rapidly, a sufficient number of cells will be obtained in a short time and so the diagnosis can be reached within two days. Complications

include bleeding, abortion (3–5 %), infection, fetomaternal haemorrhage leading to rhesus iso-immunization and limb reduction defects (controversial). Anti-D serum is given if the mother is rhesus negative and the husband is rhesus positive. The procedure allows termination of pregnancy in the first trimester if there is fetal anomaly.

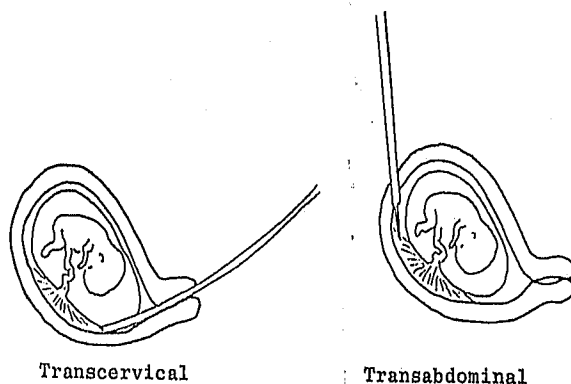


Fig. 118. Chorionic villus biopsy

2. Embryoscopy

An endoscope (embryoscope 1.7 mm in diameter) is inserted through the cervix under ultrasound guide to visualize the embryo to detect facial and limb abnormalities in the first trimester of pregnancy. The method is mainly used to confirm anomalies diagnosed by ultrasound before elective termination of pregnancy. Complications include bleeding, abortion, and infection.

3. Amniocentesis: See page (8).

4. Cordocentesis (Fetopuncture)

It is performed in selected cases from 15 weeks of pregnancy and thereafter. Under ultrasound control, using local anaesthesia, a sterile needle is passed through the abdominal wall into the umbilical vein (at the placental insertion). Fetal blood is withdrawn and examined to diagnose chromosomal abnormalities, fetal anaemia in rhesus iso-immunization and fetal infection as in case of toxoplasmosis, rubella and cytomegalovirus infection (specific antibodies are detected by radioimmunoassay) and blood factor abnormalities as haemophilia. Fetal death rate is about 1% and fetomaternal haemorrhage may occur.

5. Biopsy from Fetal Skin and Liver

Under ultrasound guide, a needle is passed to take a biopsy from fetal skin or liver. The fetoscope can be used for the same purpose.

6. Fetoscopy

The fetoscope is a thin tube (1.7 mm in diameter) which is passed through the abdominal wall under local anaesthesia to inspect the fetus, to take a sample of cord blood and to take biopsy from fetal skin and liver. The fetoscope has been replaced by passing a needle under ultrasound control.

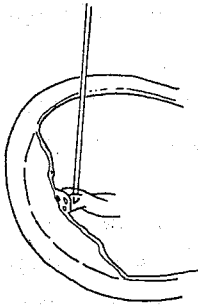


Fig.119. Cordocentesis

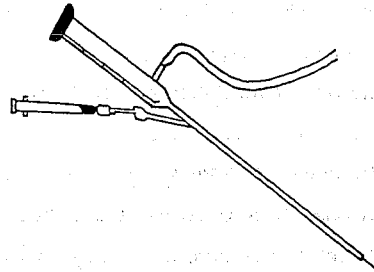


Fig. 120. Fetoscope

7. Sonography

(a) A routine ultrasound scan at 16–18 weeks or later can diagnose many of the congenital fetal abnormalities. Some abnormalities require serial ultrasound examination. It is a safe noninvasive technique; (b) echocardiography at 20–24 weeks diagnoses congenital heart disease; (c) a screening test for Down syndrome is to measure the nuchal translucency. The risk of the syndrome is 75–80% if the nuchal translucency is more than 3 mm between 10 and 14 weeks of pregnancy and more than 5 mm thereafter. The increased thickness of nuchal translucency is due to lymph collection in the subcutaneous tissue underneath the nuchal skin. Another finding is absence of the nasal bone.

8. Magnetic Resonance Imaging (MRI)

It may be indicated in some cases of congenital anomalies as it gives better information than sonography. It is costly and not done on routine basis.

9. Measurement of Alpha-fetoprotein in Maternal Serum

Done at 16–18 weeks. It is a fetal protein formed mainly by fetal liver. It is not formed by normal adult tissues. It enters the amniotic fluid and maternal blood stream. High or low values can occur with congenital fetal anomalies which must be confirmed by ultrasound or amniocentesis (high in neural tube defects as anencephaly and low in Down syndrome).

10. Screening for Down Syndrome

- a) *The double screen test.* Done between 10 and 14 weeks of pregnancy. The test is positive for Down syndrome if maternal serum shows a low level of pregnancy-associated plasma protein-A (or alpha-fetoprotein) and a high level of human chorionic gonadotrophin (HCG).
- b) *The triple screen test.* Done between 15 and 16 weeks of pregnancy (second trimester). The test is positive if maternal serum shows low levels of alpha-fetoprotein, and unconjugated oestriol, and a high level of HCG.
- c) *The quadruple screen test.* Done between 15 and 16 weeks of pregnancy. Inhibin is measured in addition to the previous 3 markers, or is measured alone. Inhibin level in maternal serum is elevated in Down syndrome.

If one of these screening tests is positive, the risk of Down syndrome is 60-75%.

- d) *Ultrasound measurement of nuchal translucency* (see above). If measurement of nuchal translucency is combined with a biochemical marker, the syndrome is detected in 90%.

A screen test is done for women below 35 years. If the test is positive, the diagnosis of Down syndrome must be confirmed by CVB, or amniocentesis according to the duration of pregnancy. If the woman is 35 years or older, CVB, or amniocentesis is directly performed without doing a screening test. All screening tests have a false positive result of 5%.

11. Preimplantation (Preconceptional) Genetic Diagnosis

In case of In Vitro Fertilization and Embryo Transfer, one or two cells (blastomeres) are aspirated from the 6, 8, or 10 cell embryo for genetic studies. Only normal embryos are transferred to the uterus. A hole is made in the zona pellucida of the morula using chemical solutions as Tyrodes solution or laser beam to aspirate the blastomeres.

12. Isolation and Analysis of Fetal Cells in Maternal Blood

These cells are trophoblastic cells, fetal lymphocytes, and nucleated fetal red cells (erythroblasts).

II. INVESTIGATIONS TO ASSESS FETAL GROWTH AND WELL-BEING

Done in late pregnancy and include:

1. Biochemical Tests

These include estimation of plasma or urinary oestriol by radioimmunoassays and measurement of human placental lactogen. However, these biochemical tests are expensive, time consuming, with high false positive and false negative results and have been replaced by biophysical tests.

2. Biophysical Tests

a) Counting of Fetal Movements

The mother is asked to count the number of fetal movements for 12 hours daily. Less than 10 movements during this period indicates fetal inactivity and a nonstress test has to be done. Another method is to count the fetal movements for one hour three times daily. A count less than 3 per hour is abnormal.

b) Nonstress Test (NST)

Normally the fetal heart rate (FHR) accelerates in response to fetal movement. The FHR is continuously recorded by the external ultrasound transducer for 20 minutes. If the mother does not feel fetal movements during this period, the recording is extended for another 20 minutes after abdominal palpation or gentle movement of the fetus to awaken the sleeping fetus and stimulate fetal movement. The test is reactive when 2 or more fetal movements are accompanied by acceleration of FHR of at least 15 beats per minute and lasting at least 15 seconds in 20 minutes. The test is nonreactive if these criteria are absent after 40 minutes of observation. A reactive test indicates that the fetus will survive for one week in most but not in all cases. So to be on the safe side, the NST is done twice weekly or even daily depending upon the reason for testing. A nonreactive test is often false positive (in at least 75% of cases due to fetal sleep) and so when the test is nonreactive a contraction stress test or a biophysical profile is performed.

Note: The NST gives a false negative result in 6 per 1000, i.e., intrauterine death occurs within one week though the test is reactive. With twice weekly testing the false negative rate is 2 per 1000.

c) Contraction Stress Test (CST)

Also known as oxytocin challenge test. A small dose of oxytocin is given by intravenous infusion and gradually increased until 3 uterine contractions are produced in 10 minutes. The test is positive if late decelerations occur with more than 50% of contractions. It is negative if no late decelerations occur and equivocal if occasional late decelerations occur with less than 50% of contractions. If the test is negative, it is repeated weekly. If the test is positive, a biophysical profile is recommended as the CST may be false positive (30%). Contraindications include classical caesarean section scar, placenta praevia and threatened preterm labour as in multiple pregnancy, polyhydramnios, incompetent cervix, and premature rupture of membranes.

Nipple stimulation may be used to avoid exogenous oxytocin and uterine hyperstimulation. It also reduces the cost and testing time. The patient is asked to rub one nipple

through the clothing for 2 minutes, or until a contraction begins. She is instructed to restart after 5 minutes if the first nipple stimulation did not induce 3 contractions in 10 minutes...

Notes:

- a. The CST is done once weekly as the false negative result is only 0.4 per 1000.
- b. The NST and CST are performed using the cardiotocograph. It records FHR and uterine contractions.

d) The Biophysical Profile (BPP)

The nonstress test is combined with 4 parameters which are assessed by ultrasound. These include fetal movements, fetal tone, fetal breathing movement and amniotic fluid volume. Each parameter is given a score of 0 or 2. A score of 8 or 10 is normal, a score of 6 is equivocal and a score of 4 or less is abnormal. If the score is normal, the test is repeated weekly or twice weekly as in diabetic cases. If the test is equivocal (suspected asphyxia) terminate if the fetus is mature, otherwise repeat testing after 24 hours. If the score is abnormal, (fetal asphyxia), pregnancy is terminated whether the fetus is mature or immature. The test takes 30 minutes.

e) The Modified Biophysical Profile

It is simpler and takes less time than the biophysical profile test (only 20 minutes or less). It includes the nonstress test and the amniotic fluid index. It is done twice weekly. The test is abnormal if the nonstress test is nonreactive or the amniotic fluid index is 5 cm or less (oligohydramnios). A reactive nonstress test indirectly indicates that the fetal breathing movements, fetal movements, and fetal tone are normal.

3. Sonography

It allows estimation of gestational age, fetal weight, rate of fetal growth to exclude intrauterine growth restriction and diagnoses fetal abnormalities. It is used to carry out the biophysical profile test. By Doppler ultrasound the rate of blood flow in maternal and fetal blood vessels can be measured (uterine artery, arcuate arteries in the placental bed, umbilical artery, fetal descending aorta, internal carotid artery and cerebral arteries). This doppler ultrasound study gives idea about fetal well-being.

4. Assessment of Fetal Lung Maturity

Amniocentesis is carried out to measure the lecithin/sphingomyelin ratio (L/S ratio). A ratio of 2 or more is associated with lung maturity. Also the presence of phosphatidylglycerol indicates lung maturity and is more reliable than L/S ratio. The respiratory distress syndrome is not liable to occur if the lung is mature.

INTRAUTERINE GROWTH RESTRICTION (RETARDATION) (IUGR)

The infant is small-for-dates if the weight is below the 10th percentile for the gestational age.

INCIDENCE

3-7% of all pregnancies

AETIOLOGY

A) Maternal Causes

1. Pregnancy induced hypertension.
2. Chronic hypertension.
3. Chronic renal disease.
4. Diabetes mellitus (advanced class D diabetes with atherosclerosis of pelvic arteries).
5. Maternal hypoxia due to any cause as anaemia and congenital heart disease.
6. Severe malnutrition, also folic acid and zinc deficiency.
7. Low prepregnancy weight <45 Kg, and poor weight gain <5 Kg during pregnancy.
8. Autoimmune diseases as systemic lupus erythematosus.
9. Antiphospholipid antibody syndrome. The presence of antiphospholipid antibodies as anticardiolipin antibodies or lupus anticoagulant.
10. Smoking leading to fetal asphyxia.
11. Drugs as folic acid antagonists, narcotics, some anticonvulsants, cocaine and alcohol.
12. Past history of IUGR (commonest cause).

B) Placental Causes

1. Small placenta.
2. Placental thrombosis and infarction.
3. Separation of the placenta in placenta praevia and abruptio placentae.
4. Circumvallate placenta.
5. True knot of the cord and loops of cord around fetal neck or body (fetal asphyxia).
6. Single umbilical artery.
7. Marginal and velamentous insertion of the cord.

C) Fetal causes

1. Chromosomal abnormalities as trisomies (trisomy 18 or 21).
2. Congenital fetal malformation as anencephaly, cardiac anomalies, and Potter syndrome.

3. Fetal infections which may be bacterial (as listeriosis and tuberculosis), viral (as rubella, cytomegalovirus, human immune deficiency virus, and varicella zoster), protozoal (as toxoplasmosis and malaria) and spirochetal in case of syphilis.
4. Multiple pregnancy as the placental mass is small in relation to fetal mass and due to twin-to-twin transfusion syndrome.
5. Dwarf syndromes such as osteogenesis imperfecta.

D) Idiopathic

In 30–50% of cases, no cause is found.

TYPES

1. Symmetrical Growth Restriction (Type 1, 20–30%)

Uniform reduction in head circumference, abdominal circumference and long bones (body length). Due to affection of the fetus by a harmful agent in early pregnancy. It is due to fetal causes, and drugs taken during pregnancy.

2. Asymmetrical Growth Restriction (Type 2, 70–80%)

The growth becomes affected after the 28th week of pregnancy. The abdominal circumference fails to enlarge at the expected rate while the head circumference and long bones continue to increase at the normal rate (brain is spared while other organs as liver and subcutaneous tissue are affected). It is due to maternal and placental causes leading to decreased uteroplacental blood flow.

3. Intermediate Growth Restriction (Type 3, 5–10%)

It is a combination of types 1 and 2 IUGR. The harmful agent affects the fetus between 20 and 28 weeks of pregnancy.

FETAL COMPLICATIONS

- A) Intrauterine asphyxia and fetal distress.
- B) Intrauterine death.
- C) After delivery, the newborn is liable to have; (1) asphyxia neonatorum due to persistence of intrauterine asphyxia after delivery; (2) meconium aspiration syndrome due to gasping in the uterus in response to asphyxia; (3) hypoglycaemia due to reduced glycogen content of the liver; (4) hypocalcaemia as asphyxia leads to acidosis which causes fetal hypothyroidism; (5) hypothermia due to lack of subcutaneous fat and hypoglycaemia; (6) polycythaemia due to chronic asphyxia; (7) hyperbilirubinaemia and jaundice in the newborn (because of polycythaemia); (8) hyperphosphataemia

secondary to tissue breakdown; (9) increased incidence of sudden infant death syndrome; (10) neurological lesions including cerebral palsy.

- D) In adult life, these babies are at increased risk of high levels of serum cholesterol, hypertension, ischaemic heart disease, diabetes, and renal damage.

DIAGNOSIS

1. Past history of a growth retarded fetus (rate of recurrence is about 25%).
2. The presence of any of the aetiological factors as pre-eclampsia.
3. Clinical examination shows small-for-dates.
4. The fundal level is lower than the period of amenorrhoea.
5. The height of the fundus above the symphysis pubis. Between 20 and 36 weeks of pregnancy, the height of the fundus in centimeters equals the duration of pregnancy in weeks. A difference more than 2 cm suggests abnormal fetal growth. The fundal height has to increase 1 cm/week (McDonald measurement).
6. Ultrasonography: It measures the different fetal diameters to estimate gestational age. The ratio of the head circumference to abdominal circumference is the most reliable index and it distinguishes symmetrical from asymmetrical growth restriction. Ultrasound also diagnoses oligohydramnios which is associated with IUGR (growth retarded fetus produces less urine due to decreased plasma volume), and shows fetal malformation which may cause symmetrical growth restriction. Doppler ultrasound may show diminished, absent, or reversed diastolic blood flow in the umbilical artery.
7. In case of symmetrical IUGR the fetal chromosomes are determined by amniocentesis or cordocentesis and the mother is screened for recent infection with the TORCH group of organisms (toxoplasmosis, rubella, cytomegalovirus, and herpes virus).
8. The ponderal index (P.I.). It is determined in the neonate. IUGR is diagnosed when the P.I. is below the tenth percentile for gestational age. The index is not evaluated with the fetus in utero during pregnancy, as the crown-heel length cannot be measured.

$$P.I. = \frac{\text{Birth weight in grams} \times 100}{\text{Cube of crown-heel length in cm}}$$

MANAGEMENT

A) Prophylaxis

A small dose of Aspirin (75 mg daily) is given after the 12th week till 38th week to patients with previous history of idiopathic fetal growth restriction to prevent recurrence. Also zinc supplementation is given between 15 and 25 weeks. Cessation of smoking.



B) During Pregnancy

1. Hospitalization.
2. Treatment of the cause if detected as maternal hypertension, malnutrition, smoking, etc.
3. Bed rest in lateral position to increase uteroplacental blood flow.
4. Serial ultrasound (every 1 or 2 weeks) to monitor rate of fetal growth.
5. Measures to assess fetal well-being:
 - a) Daily count of fetal movements.
 - b) Nonstress test twice weekly and daily in severe cases.
 - c) If the nonstress test is nonreactive do contraction stress test or fetal biophysical profile.
6. Termination of pregnancy is indicated in the following situations:
 - a) If pregnancy has reached 38 weeks (mature fetus and no benefit from waiting).
 - b) Presence of fetal asphyxia (detected by cardiotocography or biophysical profile).
 - c) Cessation of head growth (biparietal diameter and head circumference). This indicates affection of the brain.

B) During Labour

1. Continuous monitoring of fetal heart rate and uterine contractions.
2. Forceps or ventouse is applied if second stage is prolonged more than 20 minutes.

C) After labour

Proper management of neonatal problems as neonatal asphyxia, hypoglycaemia, jaundice, etc. Examination of the placenta should be considered after delivery of the IUGR fetus.

Note: IUGR = small or light-for-dates or dysmature baby.

PLACENTAL INSUFFICIENCY**DEFINITION**

It is a condition in which the respiratory and nutritive functions of the placenta are reduced due to reduction in placental circulation.

AETIOLOGY

1. Maternal causes: Page 321.
2. Placental causes: Page 321.
3. Idiopathic.

DIAGNOSIS

1. The presence of any of the aetiological factors as pre-eclampsia.
2. Clinical examination shows small-for-dates.
3. The fundal level is lower than the period of amenorrhoea
4. Sonography to diagnose intrauterine growth restriction.

FETAL COMPLICATIONS

1. Intrauterine asphyxia and fetal distress.
2. Intrauterine growth restriction.
3. Intrauterine death.

After delivery the newborn is liable to have certain complications (page 322).

MANAGEMENT: Page 323.

HAEMOLYTIC DISEASE OF THE NEWBORN (ERYTHROBLASTOSIS FETALIS)

I. RHESUS-ISOIMMUNIZATION**The Rhesus Factor**

It is carried on the surface of red cells and consists of 6 antigens; C, D, E, c, d, e. Three antigens are inherited from each parent C or c, D or d, E or e. The person is rhesus-positive if the red cells carry the D antigen. The homozygous Rh-positive person is (DD) and all his offspring will be Rh-positive. The heterozygous Rh-positive person is (Dd) and his offspring may be Rh-positive or negative. About 16% of the white populations are Rh-negative.

Pathogenesis

The condition occurs when the wife is Rh-negative, the husband Rh-positive and the fetus Rh-positive like the father. Rh-positive fetal red cells may enter the maternal circulation through breaks in the placenta during pregnancy but in most cases this happens when the placenta separates at the time of delivery. The fetal red cells stimulate the production of maternal antibodies. These antibodies may cross the placenta in subsequent pregnancies and destroy the fetal red cells. So the first fetus is not affected unless the mother was previously given an Rh-positive blood transfusion. Also the second fetus is not affected in most cases (85%) because the amount of antibodies is still small.

Incidence

The number of cases is decreasing due to prophylactic use of anti-D serum.

Clinical Picture

1. *Hydrops Fetalis*

It is the severest form. There is generalized oedema of the fetus with ascites, pericardial and pleural effusion due to heart failure. The liver and spleen are greatly enlarged, due to extramedullary erythropoiesis. The fetus dies in utero or shortly after delivery if not treated because the destruction of red cells leads to hypoxia and heart failure.

2. *Icterus Gravis Neonatorum.*

It is the commonest form. Jaundice is not present at birth but appears rapidly within the first 24 hours after delivery and becomes gradually increased. The liver and spleen are greatly enlarged. If the unconjugated bilirubin level is high the pigment may deposit in the basal ganglia of the brain leading to kernicterus which is characterized by neurological manifestations as convulsions and paralysis.

3. *Haemolytic Anaemia of the Newborn*

It is the mildest form. The fetus is born pale (anaemic) or pallor may not appear except after a few days. Liver and spleen are slightly enlarged.

Diagnosis

- a) There is usually a history of previous two normal babies.
- b) The ABO and Rh group of all pregnant women should be determined at the first antenatal visit. If the wife is Rh-negative and the husband Rh-positive, the following investigations are done:
 1. The geno-type of the husband is determined (homo- or heterozygous).
 2. The presence of anti-D antibodies and their titre or concentration is determined in maternal blood and repeated every 2 weeks.
 3. Amniocentesis is performed if the antibody titre is 1:16 or greater or the concentration is 4 international unit/ml or more (the critical level for each laboratory should be determined). Amniocentesis is also indicated if there is a past history of intrauterine death or intrauterine or exchange transfusion for a previous baby. The amniotic fluid is examined for bilirubin by the spectrophotometer using the Liley chart. This gives idea about the degree of haemolysis and the severity of the disease.
 4. Cordocentesis: Fetal blood sampling is more reliable than amniocentesis to evaluate the fetal condition. Blood is obtained from the umbilical vein to determine the haemoglobin concentration, haematocrit value, blood group, direct Coombs test and bilirubin level. This is done using a needle passed under ultrasound control or using the fetoscope.

5. Ultrasound to diagnose hydrops fetalis. The fetus appears in the "Buddha" position due to enlargement of the abdomen and oedema which interfere with flexion of the limbs. There may be a "halo" around the head due to oedema of the scalp. Doppler study of cerebral arteries can detect fetal anaemia.

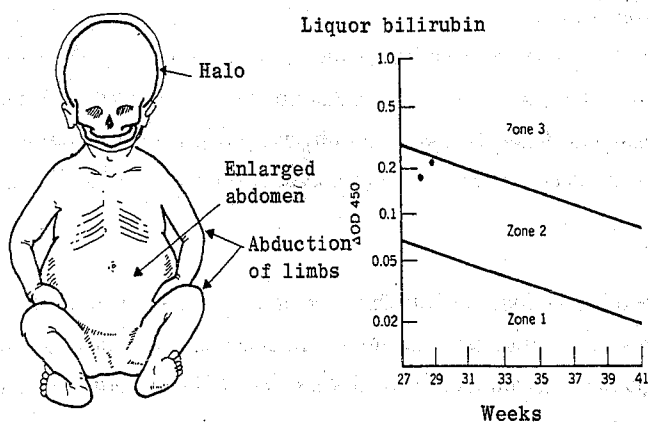


Fig. 121. Buddha position

Fig.122. Liley graph. It determines the degree of haemolysis and when to terminate pregnancy.

6. At delivery the cord blood is examined for: (a) ABO and Rh group; (b) haemoglobin per cent; (c) level of bilirubin; (d) direct Coombs test to detect the presence of antibodies on the surface of red cells.

Note: If the antibody titre remains less than 1:16, severe haemolytic disease of the newborn will not occur, and the patient is left to deliver at term.

Management

1. Prophylactic Treatment

- a) Rh-negative women should never receive Rh-positive blood transfusion.
- b) Anti-D serum is given to the Rh-negative mother provided that the fetus is Rh-positive and the maternal blood does not contain antibodies (not immunized). The dose is 300 micrograms given intramuscularly within 72 hours after delivery. However, a Kleihauer test is done 24 hours after the injection to be sure that the dose given has destroyed all fetal red cells in the maternal circulation. Another dose may be needed (20 micrograms destroys one ml of fetal red cells).
- c) Some give 300 µg at 28 and 34 weeks of pregnancy to prevent sensitization during pregnancy. However, the woman must receive another dose after delivery if indicated.

- d) The anti-D immunoglobulin is also given if there is a risk of feto-maternal haemorrhage as in case of abortion, ectopic pregnancy, evacuation of hydatidiform mole, antepartum haemorrhage, external cephalic version, amniocentesis and chorionic villus biopsy. The dose is 100 μg for first trimester abortion and 300 μg for second trimester abortion.

2. Treatment During Pregnancy

- a) *Induction of labour*: To avoid intrauterine death. It is performed at 32 weeks and onwards. Before this time the fetus is too premature to survive after delivery. The proper time of induction depends upon the bilirubin content of the amniotic fluid using Liley curve or the haemoglobin concentration or haematocrit value of fetal blood using cordocentesis.

Liley Curve

The amniotic fluid is examined for bilirubin content using the spectrophotometer.

1. Zone I. Indicates no or mild fetal anaemia. Amniocentesis is repeated within 4 weeks and pregnancy is terminated at 38-40 weeks. Fetus is monitored by nonstress test and sonography.
 2. Zone II. Moderate fetal anaemia. Amniocentesis is repeated within 1 or 2 weeks and pregnancy is terminated at 32-37 weeks. Fetus is monitored as above.
 3. Zone III. Severe fetal anaemia. The fetus is delivered immediately, and if gestational age is less than 32 weeks, intrauterine fetal transfusion is carried out.
- b) *Intrauterine fetal transfusion*: Indicated if induction of labour is needed before 32 weeks (between 16 and 32 weeks). The aim is to prolong pregnancy until the fetus is mature enough to survive after delivery. Group O, Rh-negative blood is injected by a needle passed trans-abdominally into the fetal peritoneal cavity (under ultrasound guide). Some inject the blood into the umbilical vein or inferior vena cava or a cardiac ventricle. The procedure can be repeated after 2 or 3 weeks. Ten ml of blood are given for each week after the 20th week of gestation. Thus at 30 weeks, we transfuse 100 ml (10 $\times$ 10).
- c) *Maternal plasmapheresis*: To wash antibodies from maternal blood. Indicated for severe cases between 12 and 16 weeks of pregnancy when intrauterine transfusion cannot be carried out. It is performed once or twice weekly, depending upon the anti-D titre. The treatment is costly and time consuming.

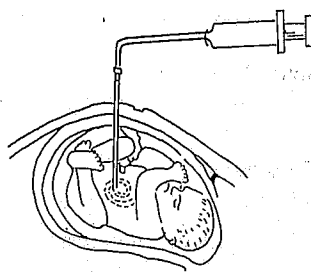


Fig. 123. Intrauterine transfusion

3. Management of the Newborn

The aim is to correct anaemia and prevent kernicterus. After delivery, the cord is immediately clamped to avoid further passage of antibodies from the placenta. The cord is divided 3 inches from the umbilicus to facilitate exchange transfusion if indicated. The cord blood is examined as mentioned before.

- a) *Exchange transfusion*: It is indicated if the unconjugated serum bilirubin reaches a dangerous level which varies according to the weight of the newborn. For example, the dangerous level is 10 mg% if the weight is 1000 grams and 20 mg% if the weight is above 2500 grams. Ten to twenty ml of blood are withdrawn from the umbilical vein and a similar amount is injected. The amount of blood needed is 160 ml/kg body weight. This allows exchange of 90% of fetal blood. The aims of the procedure are; (a) correction of anaemia; (b) removal of bilirubin; (c) removal of antibodies; (d) supply of Rh-negative cells which will not haemolyse. Exchange transfusion may be repeated if necessary.
- b) *Phototherapy*: It is used to prevent the rise in serum bilirubin. Blue or blue-green light converts the insoluble unconjugated bilirubin into a soluble form which is rapidly excreted in bile and urine without conjugation in the liver. The eyes are covered to be protected from light to avoid retinal damage. With phototherapy it is rarely necessary to perform an exchange transfusion.

II. ABO-ISOIMMUNIZATION

Erythroblastosis fetalis may occur if the mother is group O and the fetus is group A or B like the father. The group O mother has naturally occurring anti A and Anti B antibodies. Most of these antibodies have a large molecule (IgM) which cannot cross the placenta. However, some of these antibodies have a small molecule (IgG) and can cross the placenta and destroy fetal cells of group A or B. So haemolytic disease from ABO incompatibility may occur during the first pregnancy as the maternal antibodies are already present. Jaundice is usually mild because the antibodies react with the antigen A or B which is present in the red cells as well as in other fetal tissues and fluids.

III. ISO-IMMUNIZATION CAUSED BY OTHER ANTIGENS

About 700 red cell antigens exist. These can stimulate production of antibodies. However, few of these antibodies can cause significant haemolysis of red cells. Examples include anti-C, anti-E, anti-Kell and anti-kidd antibodies. In fact, 98% of cases of haemolytic disease of the newborn are due to Rh and ABO incompatibility.

Notes:

1. Kernicterus develops when the serum level of unconjugated bilirubin exceeds 20 mg%. However, it can develop at a lower concentration in the premature infant.

2. The maternal antibodies destroy the fetal red cells leading to anaemia. The fetus manufactures red cells particularly erythroblasts (primitive red cells) at rapid rate hence the name erythroblastosis fetalis.
3. Jaundice is not present at birth because the placenta removes the fetal bilirubin and excretes it into maternal circulation.
4. The anti-D serum destroys the fetal red cells which enter the maternal circulation before they stimulate production of antibodies.
5. Direct Coombs test detects the presence of antibodies on the surface of fetal red cells. The indirect Coombs test detects antibodies in maternal plasma.
6. The immunoglobulin M (IgM) is a macroglobulin with a large molecular size and does not cross the placenta, while the immunoglobulin G (IgG) with a small molecular size crosses the placenta and affects the fetus.
7. The rate of sensitization is very rare during pregnancy (1-2%) but 15-20% after delivery. With prophylactic immunization at 28 and 34 weeks gestation the rate of sensitization during pregnancy drops to 0.1%.
8. Kleihauer test. Fetal red cells remain stable in an acid pH (3.2), whereas adult Hb is eluded from the maternal red cells (ghost cells).
9. Prophylactic anti-D serum is also given in cases of abortion, ectopic pregnancy, hydatidiform mole (trophoblast carries the rhesus antigen), intrauterine death (placental integrity is disturbed), antepartum haemorrhage, external cephalic version, amniocentesis, and chorionic villus biopsy. The dose is 300 µg, however, it is 100 µg in case of first trimester abortion.
10. Normal fetal Hb is 16-18 g/dl. Anaemia is mild if Hb is 12-16 g/dl, moderate if it is 8-12 g/dl, and severe if less than 8 g/dl.
11. After delivery the cord is not milked and immediately clamped to avoid further passage of antibodies to the fetus. The cord is divided 3 inches from the umbilicus to facilitate exchange transfusion when needed.

HYDROPS FETALIS

There is generalized oedema of the fetus with ascites and pleural effusion.

AETIOLOGY

A) Immune hydrops fetalis due to ABO or Rhesus isoimmunization.

B) Nonimmune hydrops fetalis has many causes:

1. Cardiovascular as congenital heart disease and arrhythmias.
2. Pulmonary as cystic adenoma of the lung.
3. Gastrointestinal as malrotation of intestine.
4. Hepatic as polycystic disease of the liver and liver cirrhosis.
5. Renal as congenital nephrosis.
6. Placental as true knot of the cord.
7. Chromosomal as Down syndrome.
8. Infections as toxoplasmosis, rubella, cytomegalovirus, and syphilis.
9. Haematological as alpha-thalassemia, portal or caval thrombosis.
10. Twin-to-twin transfusion syndrome.

11. Medications. Indomethacin (Indocid) used to stop preterm labour may lead to premature closure of the ductus arteriosus, pulmonary hypertension, heart failure and hydrops fetalis.
12. Idiopathic.

MIRROR SYNDROME

In some cases of hydrops fetalis, the mother may develop hydrops like the fetus. The aetiology is not clear, and may be related to the production of vasoactive substances by the placenta. The mother improves only after expulsion of the fetus.

JAUNDICE IN THE NEWBORN

AETIOLOGY

1. Enzyme Deficiency

- a) Physiological jaundice. Due to immaturity of the liver and deficiency of glucuronyl transferase enzyme.
- b) Glucose-6-phosphate dehydrogenase deficiency. The enzyme is essential for the survival of red cells. Deficiency causes their spontaneous destruction.

2. Haemolytic

- a) Haemolytic disease of the newborn (erythroblastosis fetalis).
- b) Congenital spherocytic anaemia (acholuric jaundice).

3. Obstructive

Congenital obstruction of bile ducts. Jaundice appears at the end of first week of life or later. Pale stools and dark urine.

4. Infective;

- a) Congenital syphilis; causing liver cirrhosis.
- b) Viral; hepatitis A and B, herpes simplex and cytomegalovirus.
- c) Protozoal due to toxoplasmosis.
- d) Bacterial; infection of the cord may cause portal pyaemia.

5. Absorption of a Cephalhaematoma

6. Drugs

Bilirubin circulates in the blood attached to albumin. Drugs as vitamin K analogues except vitamin K₁, salicylates, sulphonamides, diazepam (Valium), and oxytocin compete with bilirubin for binding with albumin leading to hyperbilirubinaemia and jaundice.

7. Congenital Hypothyroidism.

PHYSIOLOGICAL JAUNDICE (ICTERUS SIMPLEX)

It appears 2 days or more after delivery. It is usually slight and transient. The liver and spleen are normal. Coombs test is negative, it is due to immaturity of the liver so the enzyme which conjugates bilirubin with glucuronic acid is deficient (glucuronyl transferase enzyme). It tends to be severe and prolonged in the preterm baby.

CONGENITAL FETAL MALFORMATION

INCIDENCE

The incidence of major congenital malformations is 2-3%. If minor malformations are included such as skin tags, extra digit, accessory nipples and malformed ears the rate reaches 7 to 10%.

A major malformation is defined as one that is incompatible with life, such as anencephaly, or one requiring major surgery for correction, such as congenital heart disease or cleft palate, or one producing major dysfunction, e.g., mental retardation.

AETIOLOGY

A) Genetic (Hereditary) Factors due to abnormal chromosomes or abnormal genes as in case of microcephaly and harelip (about 25%).

B) Environmental Factors

1. Irradiation especially in early pregnancy.
2. Severe dietetic deficiency, and zinc deficiency.
3. Viral diseases as German measles if acquired during the first 16 weeks of pregnancy.
4. Human toxoplasmosis.
5. Endocrine disturbances as in diabetes mellitus.
6. Fetal hypoxia resulting from maternal diseases as congenital heart disease and placental separation in placenta praevia.
7. Mechanical: Oligohydramnios may cause fetal deformities as club foot.
8. Age: Pregnancy in the extremes of age (below 20 and above 35) increases the incidence of malformation. The incidence of mongolism increases above the age of 35 years.
9. Cocaine and alcohol.

C) Drugs

Certain drugs may lead to congenital anomalies as thalidomide which gave rise to limb deformities as amelia. Antithyroid drugs may cause congenital goitre. Progestogens may

cause masculinization of the female fetus with enlargement of the clitoris and fusion of the labia minora. In general drugs may lead to congenital anomalies if given during the first ten weeks of pregnancy which is the period of organogenesis. Drugs are responsible for about 2–3% of congenital anomalies.

D) Idiopathic

The aetiology is unknown in about 65% of cases.

PRENATAL DIAGNOSIS OF CONGENITAL ABNORMALITIES

I. History

1) Maternal age 35 years or greater; (2) parental consanguinity; (3) ethnic group; (4) positive family history; (5) previous obstetric history; (6) maternal illness or medication.

II. Investigations

1) Chorionic villus biopsy; (2) embryoscopy; (3) amniocentesis; (4) cordocentesis; (5) biopsy from fetal skin and liver; (6) fetoscopy; (7) sonography; (8) magnetic resonance imaging; (9) measurement of alpha feto-protein in maternal serum; (10) screening for Down syndrome; (11) preimplantation diagnosis.

These methods are discussed in detail with "Assessment of Fetal State in Pregnancy".

FETAL THERAPY

I. MEASURES TO REDUCE CONGENITAL FETAL MALFORMATIONS

1. Folic acid supplementation to prevent neural tube defects. See diet in pregnancy.
2. Control of blood sugar in diabetic patients by diet and treatment before conception and during pregnancy.
3. Women with phenylketonuria should be kept on a diet low in phenylalanine to prevent congenital anomalies and mental retardation in the newborn.
4. Oral hypoglycaemic agents and oral anticoagulants are stopped when pregnancy is established.
5. Mega doses of vitamin A and D are avoided during pregnancy.
6. Isotretinoin (Accutane) is a vitamin A derivative used for treatment of acne vulgaris. It is highly teratogenic and should be stopped before conception.

II. DRUGS USED FOR FETAL THERAPY

1. Low-dose aspirin, 75 mg daily. It is used for prevention of pre-eclampsia, intrauterine growth restriction, and repeated fetal loss due to autoimmune diseases as systemic

lupus erythematosus and antiphospholipid antibody syndrome. See drugs during pregnancy.

2. Zinc supplementation is given between 15 to 25 weeks of pregnancy to women with previous history of idiopathic intrauterine growth restriction to prevent recurrence.
3. Dexamethasone or betamethasone. It is used to stimulate lung maturity in preterm labour before 34 weeks. The dose is 12 mg IM which is repeated after 12 hours. Its effect starts one day (24 hours) after injection and lasts for one week and may be repeated.
4. Prostaglandin inhibitors. The most commonly used is indomethacin (Indocid). It is used to inhibit preterm labour and to treat chronic polyhydramnios. See drugs during pregnancy.
5. Fetal arrhythmias. Supraventricular tachycardia is treated by giving the mother digoxin (0.5-0.75 mg orally daily) or other antiarrhythmic agents as propranolol. However, if the fetus is mature (38 weeks or more), induce labour and treat the infant in the nursery.
6. Fetal complete heart block. This may occur with connective tissue diseases as systemic lupus erythematosus. Glucocorticoids can help in patients with antinuclear antibodies.
7. Thyroxine. Fetal hypothyroidism and goitre may occur in a woman with Graves disease receiving large doses of propylthiouracil. Fetal goitre can be detected by ultrasound. In this case, a fetal blood sample is taken by cordocentesis at 35 weeks, and tested for serum thyroxine level. If it is low, thyroxine is injected into the amniotic sac at 35, 36, and 37 weeks (250 micrograms) to decrease the size of the goitre.
8. Congenital adrenal hyperplasia. When diagnosed by chorionic villus biopsy (deficiency of 21-hydroxylase enzyme), the mother is given dexamethasone before the 10th week of pregnancy and continued till delivery. This reduces masculinization of the fetal external genitalia.

III. SURGICAL TREATMENT IN UTERO

A) Closed Fetal Surgery

Surgery is done under ultrasound guide and includes the following:

1. Intrauterine fetal transfusion in case of haemolytic disease of the newborn (erythroblastosis fetalis): See haemolytic disease of the newborn for indication and technique.
2. Bilateral hydronephrosis due to posterior urethral valve. Under ultrasound guide, a special catheter is inserted inside the urinary bladder, and its other end is left in the amniotic sac. This vesico-amniotic shunt causes decompression of the kidneys and prevents atrophy of renal tissue.

3. Hydrocephalus. Cephalocentesis is performed or a ventriculo-amniotic shunt is done to drain the cerebrospinal fluid into the amniotic sac to prevent compression and atrophy of brain tissue.
4. Hydrothorax. This can lead to pulmonary hypoplasia. A thoraco-amniotic shunt may be tried.
5. Pulmonary cyst. In cystic adenomatous malformation a cysto-amniotic shunt can be done.
6. Twin-to-twin transfusion syndrome. One of the following lines of treatment may be tried to improve the prognosis:
 - a. Repeated amniocentesis of the polyhydramnios sac. This reduces the amniotic fluid pressure and placental compression, and leads to increased placental blood flow.
 - b. Laser coagulation of the anastomosing vessels on the surface of the single placenta using the fetoscope. YAG laser is used. If the anastomosing vessels are deep in the placenta, they will not be coagulated leading to a poor result.
 - c. Selective feticide of one twin using potassium chloride injected into the fetal thorax or heart. May be done if the condition is diagnosed early in pregnancy before 20 weeks.
 - d. Maternal treatment with digoxin (to prevent fetal heart failure), and indomethacin (Indocid) to treat polyhydramnios associated with the large fetus.
7. Fetal cystoscopy with laser ablation of posterior urethral valve.

B) Open Fetal Surgery

It is used for the treatment of:

1. Congenital cystic adenomatous malformations. The cystic lung mass is resected.
2. Congenital diaphragmatic hernia.
3. Urinary tract obstruction.
4. Sacrococcygeal teratoma by excision. It is the most common tumour of the newborn. Incidence 1 in 35000 live births.

Technique

Laparotomy, a trocar is placed inside the uterus and some amniotic fluid is withdrawn. The uterus and fetal membranes are incised and the membranes are attached to the edges of the uterine incision. The fetal parts to be operated upon are brought out of the uterine incision and the fetal surgery is performed. Then the uterine wound including the membranes is closed. Fibrin glue may be applied to the incision to decrease amniotic fluid leakage. Warm Ringer lactate solution with an antibiotic (500 mg vancomycin) is used to replace the amniotic fluid.

DRUGS DURING PREGNANCY AND LACTATION

I. DURING PREGNANCY

Most of the drugs have a molecular weight less than 250 and can cross the placenta. Drugs with a molecular weight more than 600 as insulin and heparin cannot cross the placenta. Drugs are responsible for 2 to 3% of congenital fetal anomalies.

There is a marked species specificity in drug teratogenesis. For example corticosteroids produce cleft palate in certain species of mice but not in humans.

The teratogenic period extends from the 4th to the 10th week of pregnancy. During this period organs are formed and a teratogen can cause malformations. However, some organs as the brain continue to develop during the second, and third trimesters of pregnancy, so we must be careful in using drugs during the first, as well as the second and third trimesters of pregnancy. The fetal alcohol syndrome can still occur when the mother starts to drink in late pregnancy. In some cases the effects of drugs appear several years after delivery.

In 1980 the American Food and Drug Administration (FDA) listed the following 5 "pregnancy categories":

- A. Well-controlled studies in pregnant women have failed to show fetal risk.
- B. Animal studies show fetal risk, but human studies don't, or animal studies are negative, but human studies are inadequate.
- C. Animal studies show fetal risk, but human studies are inadequate, or both animal and human studies are not available. A risk cannot be excluded.
- D. The drug is teratogenic in humans, or carries fetal risk, but the benefit outweighs the risk, e.g., serious diseases, or life-threatening conditions for which safer drugs are ineffective, or cannot be used.
- X. The drug is teratogenic in humans, or carries fetal risk, but the risk outweighs the benefit. These drugs are contraindicated in pregnancy.

Analgesics

1. *Aspirin*. Low-dose aspirin, 75 mg daily, is used to prevent pre-eclampsia, and recurrence of intrauterine growth restriction, to treat fetal loss due to autoimmune diseases as systemic lupus erythematosus and the antiphospholipid antibody syndrome. The drug is not teratogenic. It is antiprostaglandin, so inhibits uterine contractions and prolongs pregnancy. It decreases platelet aggregation and prolongs the bleeding time. It causes placental abruption and postpartum haemorrhage. It causes platelet dysfunction in the newborn and may lead to neonatal haemorrhage. It may cause hyperbilirubinaemia and neonatal jaundice as it competes with bilirubin for binding with albumin.

2. *Acetaminophen (Tylenol)*. It does not increase the bleeding time and does not cause platelet dysfunction in the newborn. It is safer than aspirin for mild analgesia.
3. *Nonsteroidal anti-inflammatory agents*. Not teratogenic. The one commonly used is indomethacin (Indocid). It is used to inhibit uterine contractions in preterm labour and to treat chronic polyhydramnios (see tocolytic drugs).
4. *Codeine*. It may cause maternal addiction and newborn withdrawal symptoms if used in excess.

Alcohol

The fetal alcohol syndrome (FAS) occurs in the offspring of alcoholic mothers. A safe level of alcohol intake has not been determined. The syndrome occurs in up to 30% of the newborns. The syndrome includes at least one of the following 3 features:

- a. Growth retardation before and/or after birth;
- b. Characteristic craniofacial anomalies and;
- c. Mental retardation.

Antibiotics and Anti-infective Agents

1. *Penicillin, ampicillin, amoxycillin, and erythromycin* are safe drugs. They are the first-line therapy when infection is treated in the first trimester of pregnancy. Erythromycin crosses the placenta poorly and so if given for treatment of syphilis in pregnancy because of allergy to penicillin, the newborn should be treated with penicillin.
2. *Sulphonamides*. Avoided in the third trimester of pregnancy as the drugs compete with bilirubin for binding sites on albumin leading to fetal hyperbilirubinaemia and neonatal jaundice. They are also avoided in women having glucose-6-phosphate dehydrogenase deficiency to avoid red cell haemolysis which is dose related.
3. *Nitrofurantoin (Macrochantin)*. Used for the treatment of asymptomatic bacteriuria and lower urinary tract infection. Avoided in women having glucose-6-phosphate dehydrogenase deficiency to avoid red cell haemolysis. It also causes haemolysis in the newborn deficient in this enzyme.
4. *Tetracyclines*. They combine with calcium in bone and teeth leading to yellow or brown discolouration of deciduous teeth, hypoplasia of the enamel, and delayed growth of bone. This only happens if the drug is taken after the 4th month of pregnancy when calcium starts to deposit in bone and teeth, so the drug is safe in the first trimester. Tetracyclines are hepatotoxic if given in large doses.
5. *Cephalosporins*. No teratogenic effects have been associated with their use.
6. *Aminoglycosides*. Streptomycin and kanamycin cause 8th nerve damage and congenital deafness (10-15%).

7. *Antituberculous drugs*. Isonicotinic acid hydrazide, para-aminosalicylic acid, rifampin, and ethambutol are not teratogenic.
8. *Metronidazole*. Studies did not show teratogenic or carcinogenic effects when given in early or late pregnancy.
9. *Acyclovir (Zovirax)*. For treatment of genital herpes. Not teratogenic.
10. *Antifungal agents*. They are not teratogenic.

Anticoagulants

1. *Oral anticoagulants* as warfarin cause abortion, intrauterine growth restriction, intrauterine death, maternal and fetal haemorrhage, and congenital fetal anomalies known as fetal warfarin syndrome with characteristic craniofacial anomalies.
2. *Heparin*. It does not cross the placenta, and is not secreted in milk. It is the drug of choice for anticoagulation in pregnancy as it does not affect the fetus. Maternal complications include haemorrhage, thrombocytopenia, alopecia, allergic reactions, hypotension, and osteoporosis if large doses more than 15000 units daily are given for 6 months.

Anticonvulsants

Hydantoin anticonvulsants as phenytoin cause fetal hydantoin syndrome in up to 30% of cases. It is characterized by mental retardation, and characteristic craniofacial anomalies. Phenytoin reduces the absorption of folate and 5 mg folic acid should be taken daily to prevent neural tube defects. The benefits of anticonvulsant treatment outweigh the risks of discontinuation of the drug. However, in petit mal epilepsy, which is a mild disease, no drugs are given as the risks outweigh the benefits.

Antihypertensive Drugs

The angiotensin-converting enzyme inhibitors, such as enalapril are contraindicated in pregnancy, as they cause fetal renal failure, oligohydramnios, neonatal anuria, craniofacial anomalies, lung hypoplasia, and limb contractures. Other antihypertensives are not teratogenic.

Cytotoxic Drugs

Methotrexate and aminopterin are folic acid antagonists. They lead to abortion, fetal death, intrauterine growth restriction, and multiple congenital anomalies.

Immunosuppressant Drugs

1. Azothioprine (Imuran) used by patients with renal transplant is not teratogenic. Maternal complications include infection, bone marrow depression, and hepatotoxicity.

2. Cyclosporine is not teratogenic. It may lead to intrauterine growth restriction and preterm labour.

Antithyroid Drugs

These cross the placenta and may cause fetal hypothyroidism and goitre. Propylthiouracil is the drug of choice during pregnancy as carbimazole and methimazole may lead to aplasia cutis (rare) in the fetus.

Iodide

Iodide, such as found in potassium iodide expectorant may produce fetal hypothyroidism and goitre. Iodide decreases the release of thyroid hormones.

Steroid Hormones

Women may continue to take contraceptive tablets unaware that they are pregnant. Recent studies have failed to show any teratogenic effect for these pills. Progestogens taken during the first trimester of pregnancy may cause enlarged clitoris and fused labia minora (1-2%). Danazol produces the same effects.

Vitamins

1. Folate deficiency is associated with increased incidence of neural tube defects as anencephaly and spina bifida.
2. Mega doses of vitamin A more than 15000 i.u daily are teratogenic. A dose between 10000 and 15000 i.u daily may be teratogenic. A dose below 10000 i.u daily is safe. Isotretinoin (Accutane) is a vitamin A derivative used for the treatment of acne vulgaris. It is highly teratogenic causing multiple congenital anomalies in 25% of cases and additional 25% of the newborns show mental retardation.
3. Mega doses of vitamin D are teratogenic.

Lithium

It is teratogenic. It may lead to cardiac anomalies, fetal hypothyroidism and goitre, and polyhydramnios due to nephrogenic diabetes insipidus. Discontinuation leads to relapse of mental illness in 70% of cases within one year, so it is given during pregnancy but sonography and echocardiography must be done.

Notes:

- Some drugs have split designations, e.g. B/D. This means the drug is not teratogenic but gives fetal problems when given before delivery as sulphonamides and indomethacin.
- Prednisone and prednisolone are inactivated by the placenta and the concentration of active compound in the fetus is less than 10% of that in the mother. When steroid effects are desired in the fetus for example, to accelerate lung maturity, dexamethasone or betamethasone is given, as these are minimally inactivated by the placenta.

II. DURING LACTATION

Most of the drugs are safe during lactation, as small amounts appear in breast milk, approximately 1-2% of the maternal dose.

Drugs Contraindicated During Lactation

1. *Antithyroid drugs*. They cause fetal hypothyroidism and goitre. However, if propylthiouracil is given in a small dose less than 200 mg daily, breast-feeding can continue with close supervision of the infant for hypothyroidism.
2. *Alcohol*. Alcohol levels in breast milk are similar to those in maternal plasma. Intoxication of the infant may occur. Chronic alcoholism can affect motor but not mental development. The odour of milk is changed and the infant may refuse breast-feeding. Alcohol also reduces oxytocin release and milk secretion.
3. *Amphetamines*.
4. *Bromocriptine*. It inhibits milk secretion.
5. *Cytotoxic and immunosuppressant drugs*.
6. *Combined contraceptive tablets* as they suppress milk secretion, but have no effect on the fetus.
7. *Ergotamine*. In doses to treat migraine can cause nausea, vomiting, and convulsions in the infant. Short ergot therapy to stimulate uterine contractions in the postpartum period is not a contraindication.
8. *Lithium*. It causes fetal hypothyroidism, goitre, and nephrogenic diabetes insipidus.
9. *Metronidazole* if given in a single large dose (2 g). Lactation is stopped for 12-24 hours.
10. *Radioactive compounds* used for diagnostic purposes. Breast-feeding is stopped for a period depending on the radioactive substance used, e.g., 4 days for radioactive sodium. The milk may be counted for radioactivity before nursing is recommended.

Note: Progestogen-only pills do not affect the quantity or quality of breast milk making them ideal in the breast-feeding mother.

IMMUNIZATION DURING PREGNANCY

TYPES OF VACCINES

A) Live Attenuated Virus Vaccines

1. *Measles*. Contraindicated during pregnancy.
2. *Rubella*. Contraindicated during pregnancy.
3. *Mumps*. Contraindicated during pregnancy.
4. *Polio myelitis*. Two types; live attenuated virus given orally (OPV) and inactivated virus (IPV) vaccine given subcutaneously. Vaccine (any type) is given to the pregnant

woman travelling to endemic areas. No risk is confirmed from the vaccine to the fetus. Also, the risk from an attenuated virus is less than that caused by the virulent virus in the endemic area.

5. *Yellow Fever*. Contraindicated except if exposure is unavoidable. Postponement of travel is preferable to vaccination, if possible. Risk from vaccine to the fetus is unknown.
6. *Varicella*. Contraindicated during pregnancy.

B) Inactivated (Killed) Virus Vaccines

These are not contraindicated during pregnancy.

1. Influenza.
2. Rabies.
3. Poliomyelitis. The IPV vaccine which is given subcutaneously.

C) Inactivated Bacterial Vaccines

These are killed bacterial vaccines and are not contraindicated during pregnancy.

1. Cholera.
2. Meningococcus.
3. Typhoid.
4. Plague.

D) Toxoids

Toxoid is a bacterial toxin that has been treated chemically to destroy its toxicity, but still capable of producing antibodies. Toxoids as tetanus and tetanus-diphtheria toxoids are not contraindicated during pregnancy.

E) Immune Globulins

These are not contraindicated during pregnancy.

- | | |
|-----------------|---------------|
| 1. Hepatitis A. | 4. Varicella. |
| 2. Hepatitis B. | 5. Tetanus. |
| 3. Measles. | 6. Rabies. |

OPERATIVE OBSTETRICS

TERMINATION OF PREGNANCY

I. INDUCTION OF ABORTION

Therapeutic abortion means termination of pregnancy before 20 weeks for a medical reason.

Indications

A) General Indications

1. Certain cases of hyperemesis gravidarum.
2. Severe cases of chronic hypertension.
3. Advanced chronic renal disease.
4. Organic heart disease if there is failure in early pregnancy or a history of failure in previous pregnancy or before pregnancy.
5. Respiratory diseases as pulmonary tuberculosis or severe bronchial asthma that cannot be controlled.
6. Breast carcinoma with pregnancy or if pregnancy occurs within two years after radical mastectomy.
7. Severe diabetes mellitus, complicated by diabetic nephritis or retinopathy or ischaemic heart disease. However, these are not absolute indications but the risks should be explained to the patient.
8. German measles acquired in the first 16 weeks of pregnancy.
9. Exposure to X-ray in early pregnancy.
10. Other rare indications as chorea gravidarum, mental disorders, epilepsy and thyrotoxicosis, if these diseases cannot be controlled during pregnancy.

B) Local Indications

1. Missed abortion.
2. Hydatidiform mole.
3. Acute polyhydramnios.
4. Carcinoma of cervix.
5. Incarcerated retroverted gravid uterus which cannot be corrected.

Methods

I. During the First 12 Weeks of Pregnancy

A) Medical methods

1. The use of RU-486 (Mifepristone).
2. The use of epostone.

B) Surgical methods

1. Cervical dilation and evacuation of uterine contents.
2. Suction evacuation. The suction cannula is available in various sizes, 6, 8, 10, 12 and 14, indicating the diameter in millimeters. The proper size is equivalent to the number of weeks of pregnancy.
3. Menstrual aspiration.

II. After 12 Weeks of Pregnancy

A) Medical methods

1. Oxytocin infusion; used in missed abortion.
2. Prostaglandins (page 158).

B) Surgical methods

1. Rupture of membranes in case of acute polyhydramnios.
2. Intra-amniotic injection of hypertonic solutions; saline 20% or urea 40%.
3. Abdominal hysterotomy.

RU-486 (Mifepristone)

It blocks progesterone receptors. It is available in 50 milligrams tablets. To induce abortion 4 tablets (200 mg) are given daily for 4 successive days (90% will abort), or a single large dose of 12 tablets (600 mg) is given in combination with a prostaglandin given IM or as vaginal tablet. This causes abortion in 99% of cases. To terminate pregnancy in the second trimester, 4 tablets are given and after 24 hours we start extra-amniotic prostaglandin infusion. It is more effective than giving prostaglandin alone.

Epostone

The drug is given orally and it inhibits the synthesis of endogenous progesterone. It causes abortion in 80% of cases, if given in the first 4 weeks of the last menstrual period.

Technique of Vaginal Evacuation

General anaesthesia or paracervical block. Lithotomy position. The vulva, vagina, and perineum are painted with an antiseptic solution, and sterile towels are applied. Bladder

evacuated by a catheter. Bimanual examination to confirm the diagnosis and to exclude any abnormality. The cervix is exposed by inserting a self-retaining posterior vaginal speculum (Auvard speculum). The cervix is grasped by ring forceps or volsellum. A uterine sound is passed to determine the length and direction of uterus. The cervix is gradually dilated until the ovum or ring forceps can be introduced (dilatation to 12 mm is sufficient in most cases). The products of conception are removed by the ovum or ring forceps. The forceps is introduced while closed into the uterus, it is then opened and closed to grasp the contents, and rotated 90° before withdrawal to be sure that the uterine wall is not grasped. The process is repeated till the uterus is felt empty. The uterine cavity is then curetted by a sharp curette to remove the decidua, ergometrine is given I.V. during the operation to harden the uterus to minimize bleeding and protect against perforation.

In case of suction evacuation, the same steps are followed. Then the cervix is dilated using a Hegar dilator, whose number equals the duration of pregnancy in weeks, so for 8 weeks pregnancy, we dilate to no. 8 Hegar, i.e. to 8 mm. A suction cannula of the same size is introduced into the uterus. The cannula is attached by a flexible tube to a suction apparatus. Suction is begun (to create a vacuum pressure of 500–600 mmHg) and the cannula is moved over the whole endometrial surface in a systematic way. Sharp uterine curettage may be done after aspiration of uterine contents. The advantages of suction evacuation are: (1) it takes a shorter time; (2) less anaesthesia is required; (3) less bleeding; (4) lower incidence of perforation; (5) less traumatic and intrauterine adhesions are less likely to occur.

After vaginal evacuation the products of conception should be examined macroscopically and microscopically.

Abnormal bleeding during or after evacuation may be due to:

1. Retained products. These should be removed and uterus curetted until a gritty sensation is heard and felt.
2. Atony of uterus. Managed by ergometrine I.V. and massage. A uterine pack may be inserted and removed after 24 hours.
3. Laceration of the cervix. Managed by suturing the tears.
4. Perforation of uterus. When this occurs during evacuation of a pregnant uterus, evacuation is completed under laparoscopic vision by a second operator, or under ultrasound guide.

Complications of Vaginal Evacuation

1. Shock. If cervix is dilated before the patient is completely anaesthetized.
2. Haemorrhage. It is due to retained products, uterine atony, perforation of uterus or cervical lacerations.

3. Infection.
4. Perforation of uterus.
5. Cervical lacerations.
6. Incompetent cervix which may lead later to habitual abortion or preterm labour.
7. Perforation of bladder and damage to intestine.
8. Intrauterine adhesions (Asherman Syndrome).
9. Anaesthetic complications.

Menstrual Aspiration

The uterine cavity is aspirated using a special cannula (Karman cannula) and a 50 ml syringe 1–3 weeks after a missed period to terminate early pregnancy.

Note: Laminaria tents are made from the stem of a special seaweed (*Laminaria Japonica* and *Laminaria Digitata*). They are available in various diameters ranging from 3 to 10 mm. They are hygroscopic, they swell and cause dilatation of cervix. If the cervix is firm and looks difficult to dilate, a tent of suitable size is inserted into the cervical canal with its tip just beyond the internal Os and left 12–24 hours before attempting evacuation. A synthetic dilator – Lamicel or Dilapan – is now available and acts more rapidly. A prostaglandin vaginal suppository or gel can also cause ripening of the cervix (inserted 12–24 hours before operation).

Intra-Amniotic Injection of Hypertonic Solutions

A needle is inserted through the abdominal wall under local anaesthesia midway between the uterine fundus and symphysis pubis and pushed into the uterine cavity. As much liquor as possible is withdrawn from the amniotic sac (ideally 100–200 ml) and replaced with an equal volume of 20% sodium chloride solution or 40% urea solution in 5% glucose. In many cases (90%), the fetus is expelled within 48 hours. If uterine contractions are weak, oxytocin infusion is started. The mode of action is unknown, but the hypertonic solution may absorb fluid from the fetal, maternal and placental tissues leading to distension of uterus and uterine contractions or the hypertonic solution leads to placental necrosis and release of prostaglandins which stimulate uterine contractions. The complications of hypertonic saline include: (1) hypernatraemia, if sodium is injected into maternal circulation; (2) hypervolaemia and heart failure; (3) water intoxication; (4) disseminated intravascular coagulation (the necrotic placenta releases thromboplastin); (5) myometrial necrosis if saline extravasates into uterine muscle; (6) peritonitis and (7) septic shock. Urea is less toxic and preferred to saline.

This amnioinfusion is rarely used nowadays because maternal deaths occurred in some cases. However, it is still used in some areas of the United States.

Hysterotomy

Abdominal hysterotomy is performed if all measures fail to induce abortion after the 12th week. Abdominal hysterotomy is a miniature caesarean section done before 20 weeks of pregnancy.

II. INDUCTION OF LABOUR

It is termination of pregnancy after the 20th week (after viability of fetus).

Indications

A) Maternal

1. Pre-eclampsia.
2. Antepartum eclampsia; if labour does not start after control of fits and the fetus is alive.
3. Chronic hypertension.
4. Chronic renal disease.
5. Certain cases of antepartum haemorrhage (placenta praevia and abruptio placentae).
6. Diabetes mellitus.
7. Premature rupture of membranes if duration of pregnancy is 36 weeks or more, and at any time if there is fetal distress, chorioamnionitis, or evidence of fetal lung maturity (presence of phosphatidylglycerol in vaginal fluid).

B) Fetal

1. Postterm pregnancy.
2. Fetal malformation as anencephaly.
3. Intrauterine growth restriction.
4. Repeated unexplained intrauterine fetal death before term.
5. Certain cases of rhesus incompatibility.
6. Placental insufficiency.
7. Unstable lie.

Methods of Induction of Labour

A) Stripping of The Membranes

The index finger is used to separate the fetal membranes (chorion and amnion) from the wall of the cervix and lower uterine segment. This stimulates release of prostaglandins from the membranes and adjacent decidua. In addition the method may cause release of maternal oxytocin from the posterior pituitary. The efficacy of this method is not proven, and it should not be used alone to induce labour. Complications include rupture of the membranes, uterine infection and bleeding from undiagnosed placenta praevia.

B) Medical Induction

1. Oxytocin Infusion

Ten units of oxytocin in 500 ml normal saline or 5% glucose in normal saline (to avoid water intoxication if glucose is used alone). Start by 2 milliunits/minute (2 drops/min) and the dose is doubled every 15 minutes but don't exceed 32 mU/min (32 drops). The aim is to produce 3 uterine contractions in 10 minutes (like contractions of normal labour lasting 40–50 seconds and producing intrauterine pressure of 50–60 mmHg). Ideally an infusion pump is used to deliver the desired amount. During induction the fetal heart rate and uterine contractions are continuously monitored. If the method fails it can be repeated after one day.

Oxytocin nasal drops or spray or buccal tablets are more convenient to the patient but the absorption is irregular and the response of uterus cannot be predicted. These methods can be used to ripen the cervix before oxytocin infusion.

10 units in 500 ml = 10,000 milliunits in 500 ml = 20 mU/ml = 20 mU/20 drops because 1 ml = 20 drops in most sets used for infusion. So one drop will contain 1 mU of oxytocin.

2. Prostaglandins (page 158).

C) Surgical Induction

It is by artificial rupture of membranes (amniotomy). It is applied in cephalic presentation when the head is engaged or at least well-applied to the pelvic inlet to avoid cord prolapse, and the cervix is ripe, that is short, soft and partially dilated.

1. Rupture of the Forewaters

It is done without anaesthesia. The index finger is introduced to separate the membranes from the lower uterine segment. The membranes are ruptured with Amnihook or a Kocher or volsellum. The fetal head is displaced upwards slightly to allow escape of as much liquor as possible. If labour does not start within 6 hours the uterus is stimulated with oxytocin drip and if labour does not start within 12 hours after rupture of membranes caesarean section is performed.

2. Rupture of the Hindwaters.

It is done under general anaesthesia using the Drew Smythe catheter. It is passed between the membranes and uterine wall to reach above the presenting part. As much liquor as possible is drained and then the catheter is removed.

Advantages

1. No risk of cord prolapse.

2. The intact bag of forewaters helps to dilate the cervix when labour starts.
3. The amount of liquor drained can be controlled, so useful in case of chronic polyhydramnios.

Disadvantages

Injury of the fetus, placenta or uterine wall and it is more difficult. We prefer rupture of the forewaters because it is easy and safe.

D) Combined Induction

1. Oxytocin drip is started and when the cervix is half dilated the membranes are ruptured or
2. Amniotomy is done and if labour does not start within 6 hours oxytocin infusion is given or it is given immediately after amniotomy.

N.B.: Oxytocin drip will induce labour in about 60% of cases and amniotomy in 80%. Combination of the two methods gives better results. Amniotomy induces labour through release of prostaglandins.

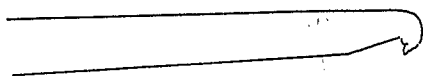


Fig.124. Amnihook

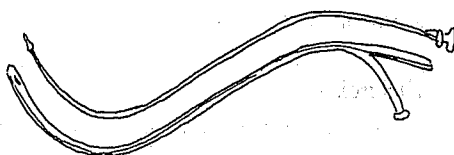


Fig.125. Drew Smythe catheter

CAESAREAN SECTION

DEFINITION

It is delivery of the fetus after 20 weeks of pregnancy (after viability) through an abdominal and uterine incision.

INCIDENCE

Varies from 10 to 25% of all deliveries. However, the rate is increasing all over the world.

INDICATIONS

A) Maternal

1. Contracted pelvis and fetopelvic disproportion (page 228).

2. Certain cases of antepartum haemorrhage:
 - a) Placenta praevia (page 246).
 - b) Abruptio placentae (page 253).
3. Abnormal uterine action during labour as high retraction ring and cervical dystocia.
4. Previous caesarean section. Caesarean section is repeated in the following conditions:
 - a) Contracted pelvis.
 - b) Previous 2 caesarean sections.
 - c) If the previous operation was in the upper segment.
 - d) If there is a history of fever in the puerperium (this suggest uterine infection and weak scar).
 - e) The presence of malpresentation as breech or transverse lie. In breech presentation intrauterine manipulations may be required to deliver the legs or arms.
 - f) If the scar is tender or there is vaginal bleeding (threatening rupture).
5. Previous gynaecological operations:
 - a) Repaired vesicovaginal fistula.
 - b) Repaired stress incontinence.
 - c) Fothergill operation.
 - d) Scar in the uterus after uteroplasty or myomectomy (when the uterine cavity was opened or myometrium extensively damaged during removal of fibroids).
6. Soft tissue obstruction:
 - a) Carcinoma of the cervix or cervical fibroid.
 - b) Tumour impacted in the pelvis, e.g. fibroid or ovarian cyst.
7. Maternal diseases:
 - a) Certain cases of diabetes mellitus.
 - b) Some cases of pre-eclampsia and eclampsia and other hypertensive disorders of pregnancy.
 - c) Infection of the genital tract with herpes virus to prevent infection of the newborn (active genital herpes with visible lesions in the genital tract unless the membranes have been ruptured for more than 4 hours).
8. Maternal distress before the cervix is fully dilated.
9. Elderly primigravida, if there is danger to the fetus from vaginal delivery.

B) Fetal

1. Large fetus.
2. Certain cases of malpresentation:
 - a) Breech (page 179).
 - b) Transverse or oblique lie (page 190).
 - c) Occipito-posterior, face or brow.

3. Fetal distress before the cervix is fully dilated.
4. Prolapsed pulsating cord before the cervix is fully dilated.
5. Repeated intrauterine fetal death in the last weeks of pregnancy.
6. Some cases of Rh-isoinmunization.
7. Postmortem caesarean section, to save the fetus within 10 minutes after death of the mother.

Note: The commonest indication is a previous caesarean section. This accounts for about 30% of all sections.

CONTRAINDICATIONS

1. Congenital fetal anomalies as hydrocephalus unless vaginal delivery is dangerous for the mother.
2. Dead fetus, however, caesarean section is performed on a dead fetus in the following conditions:
 - a) Extreme degree of pelvic contraction (true conjugate diameter is less than 6 cm).
 - b) Placenta praevia centralis.
 - c) Some cases of concealed type of placental abruption.
 - d) Soft tissue obstruction which prevents vaginal delivery as cervical fibroid.
 - e) Neglected shoulder presentation. Operation is safer for the mother than decapitation.

The above are absolute indications for caesarean section.

TIMING OF THE OPERATION

1. Elective Caesarean Section

The operation is done before onset of labour, usually one or two weeks before the expected date of delivery.

2. Selective Caesarean Section

The operation is performed after onset of labour.

TYPES OF OPERATION

1. The upper segment or classical caesarean section.
2. The lower segment caesarean section.

Technique of Lower Segment Caesarean Section

General, spinal or epidural anaesthesia, Acupuncture, or local infiltration anaesthesia as in case of congestive heart failure. Bladder is evacuated and kept empty by a Foley catheter and fetal heart sounds are auscultated. A midline subumbilical incision is made reaching the upper border of symphysis pubis. However, a transverse suprapubic incision

is preferred (Pfannenstiel incision). The peritoneal cavity is opened. Centralize the uterus by inspecting or palpating the round ligaments as the uterus is commonly dextrorotated (80%). The bladder is drawn downwards by a Doyen retractor. The loose peritoneum over the lower segment is incised transversely for about 10 cm and dissected downwards with the bladder and keep them against the symphysis pubis by the retractor. Incise the lower segment transversely for about 10 cm. The fetus is delivered. Ergometrine or oxytocin is given intravenously. The placenta and membranes are then delivered. The uterine cavity is wiped out with gauze to remove placental fragments, pieces of membranes, blood clots and vernix caseosa. The uterus is closed in 3 layers using chromic catgut or Vicryl, the muscle in 2 layers and the peritoneum as a separate coat. Clean the peritoneal cavity, close the abdomen, and finally remove blood clots from the vagina.

N.B.:

- The head is delivered during caesarean section by: (1) introducing the hand below the head and pushing it upwards and this is helped by moderate pressure on the fundus; (2) the blade of forceps which is used as a lever to extract the head; (3) application of Wrigley forceps; (4) Willett scalp forceps.
- The new trend is to close the muscle wall of the lower uterine segment in one layer using a continuous locking suture. This reduces endometritis and gives a stronger scar as closure in 2 layers may lead to ischaemia of tissues and a weaker scar. Also, the visceral and parietal peritoneum are not closed and this reduces time and money and may reduce formation of adhesions.

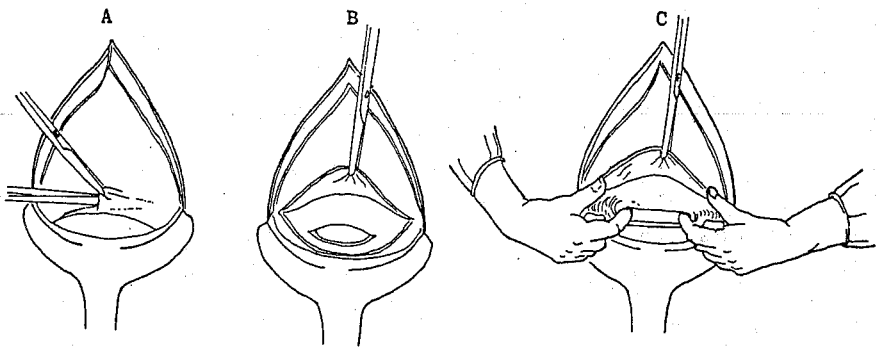


Fig. 126. Lower segment Caesarean section. (A) Loose peritoneum over the lower segment is incised; (B) a small incision is made in the lower segment; (C) Incision is enlarged by finger traction

Advantages of the Vertical Incision in the Lower Segment

1. Less bleeding because the middle line is the least vascular area of the uterus.
2. It is away from the uterine vessels and ureter which may be injured by a transverse incision.

3. Less liability to pelvic thrombosis and embolism.
4. Repair is more anatomical as the flaps are of equal thickness.

The disadvantage of this incision is that it may extend downwards into the bladder or vagina. It is indicated if there is a contraction ring or varicose veins over the lower segment to avoid the dilated vessels. This vertical incision is known as Kronig incision.

ADVANTAGES OF THE LOWER SEGMENT OVER THE UPPER SEGMENT OPERATION

<i>Lower Segment Operation</i>	<i>Upper Segment Operation</i>
1. Haemorrhage is less because: a) Lower segment is thin and less vascular. b) The incision is away from the placental site.	1. Haemorrhage is more as: a) Upper segment is thick and more vascular. b) The incision is near or on the placental site.
2. The scar is strong and less liable to rupture. The incidence of rupture in subsequent pregnancy or labour is 0.2%. The reasons are: a) The lower segment is relaxed during the puerperium allowing better healing. b) The lower segment is thin and this allows good approximation of the edges. c) The scar is away from the placental site and so it is not eroded by chorionic villi in subsequent pregnancy.	2. The scar is weak and more liable to rupture. The incidence of rupture is 2%, because; a) The upper segment is active and this interferes with healing. b) The wall is thick and so approximation is difficult. c) Erosion of the scar is liable to occur, because the placenta is normally situated in the upper uterine segment.
3. If uterine infection occurs it remains extraperitoneal and localized in the pelvis because the peritoneum is closed as a separate layer.	3. Uterine infection is liable to spread to the peritoneal cavity.
4. Less liability to paralytic ileus and acute dilatation of the stomach as the intestine is not manipulated during operation.	4. More liability to paralytic ileus and acute dilatation of the stomach.
5. Less liability to postoperative adhesions and intestinal obstruction because the site of the wound is low and covered with peritoneum.	5. Postoperative adhesions and intestinal obstruction are more frequent.
6. Mortality rate is low.	6. Slightly higher.

Advantages of Upper Segment Operation

1. It is easy, while the lower segment operation is more difficult.
2. It takes a short time. The other operation takes a longer time.

Technique of Upper Segment Caesarean Section

A midline subumbilical or a paramedian incision. A vertical incision 10 cm long is made in the upper uterine segment. After delivery of the fetus, placenta and membranes the uterus is closed in 3 layers. The first includes the deep part of the thick myometrium, the second includes the middle part and the third includes the superficial part and the adherent peritoneum.

Note: Paramedian incision is $\frac{1}{3}$ above and $\frac{2}{3}$ below the umbilicus.

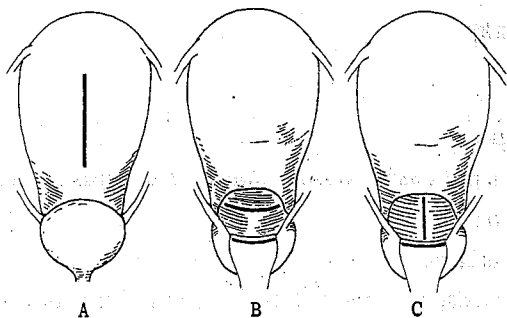


Fig. 127. (A) Vertical incision in the upper uterine segment; (B) transverse incision in the lower segment; (C) vertical incision in the lower segment.

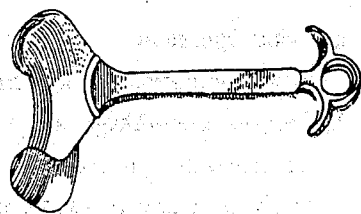


Fig. 128. Doyen retractor

Indications of Upper Segment Caesarean Section

1. If it is difficult or dangerous to reach the lower segment due to the presence of fibroids, varicose veins, or extensive adhesions.
2. Concealed type of abruptio placentae because the bad maternal condition needs rapid delivery.
3. Impacted shoulder presentation.
4. Repaired vesicovaginal fistula.
5. If caesarean hysterectomy is indicated.

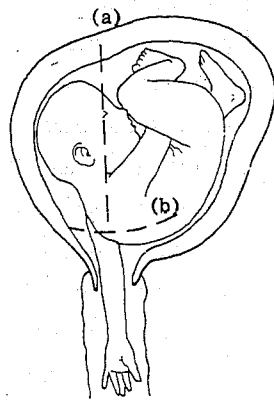


Fig. 129. Impacted shoulder
Incision (a) is more helpful than (b)

6. Postmortem caesarean section.

Caesarean Hysterectomy

Caesarean section is performed followed by removal of the uterus.

Indications

1. Uncontrollable postpartum haemorrhage.
2. Some cases of rupture of uterus.
3. Placenta accreta.
4. Fibroid uterus needing hysterectomy.
5. Some cases of operable carcinoma of cervix with pregnancy.
6. Gross uterine infection.

COMPLICATIONS OF CAESAREAN SECTION

I. During Operation

1. Shock; neurogenic or haemorrhagic.
2. Primary haemorrhage due to injury of uterine vessels, atony of the uterus and coagulation defects as in case of abruptio placentae.
3. Injury to the urinary bladder and ureters.
4. Anaesthetic complications as Mendelson syndrome due to aspiration of vomitus. The patient should fast for at least 6 hours before operation except in emergency cases. An antacid as sodium citrate (30 ml) and one Zantac tablet are given orally before operation.

II. Postoperative

1. Cardiovascular: Reactionary and secondary haemorrhage, venous thrombosis and pulmonary embolism.
2. Pulmonary: As bronchitis and pneumonia.
3. Genital tract: Infection as endometritis, parametritis and peritonitis. Endometritis is the commonest complication (5-35%).
4. Gastrointestinal tract: Acute dilatation of the stomach, paralytic ileus, later adhesions and intestinal obstruction.
5. Urinary tract: Retention of urine, infection (due to catheterization), urinary fistula and menouria syndrome.
6. Complications in the abdominal wound. Infection, burst abdomen and incisional hernia. Incisional hernia is very rare after Pfannenstiel incision (1 in 1250 cases).
7. Rupture of the uterine scar in subsequent pregnancy or labour (about 0.2% after lower segment and 2% after upper segment operation).

8. Endometriosis due to implantation of endometrial cells in the pelvis, peritoneal cavity, uterine or abdominal incision.

The mortality rate is less than one in thousand (1 in 2500 to 1 in 5000).

Notes:

- The menouria syndrome means amenorrhoea and cyclic haematuria due to the presence of a vesicouterine fistula which may follow lower segment caesarean section. The syndrome occurs if the uterine opening of the fistula is above the level of internal os of cervix.
- The average blood loss at caesarean section is about 750–1000 ml. The loss is less with epidural anaesthesia (about 500 ml). With Acupuncture plus local analgesia the blood loss is reported to be less than 250 ml.

LABOUR FOLLOWING CAESAREAN SECTION

The old rule once caesarean always caesarean has been changed to once caesarean always hospital delivery. The patient may be allowed to deliver vaginally or caesarean section is repeated. The indications for repeated section have been mentioned before. If vaginal delivery is allowed continuous electronic fetal monitoring is indicated. After delivery the uterine cavity may be explored to assess the uterine scar.

CAESAREAN STERILIZATION

Tubal sterilization is advised during the third section.

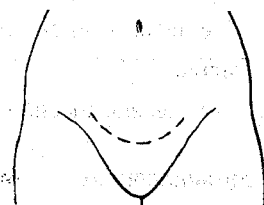
Notes:

- Most women can attempt pregnancy 3–6 months after caesarean section.
- The thickness of uterine scar can be assessed by ultrasound. If it is 4.5 mm or more after 36 weeks of pregnancy, rupture of the scar is not liable to occur.

PFANNENSTIEL INCISION

Technique

A transverse semilunar incision 10–15 cm long is made in the skin and subcutaneous fat. The incision is made in the natural crease above the pubic hairline or 2–3 cm above the symphysis pubis. The anterior rectus sheath is incised transversely. The upper and lower flaps of this fascia are separated from the underlying rectus muscles by blunt dissection. The rectus muscles are separated in the midline to expose the underlying parietal peritoneum which is incised vertically to open the peritoneal cavity.



Advantages

(1) Less postoperative pain; (2) less medication for pain; (3) early ambulation; (4) less postoperative pulmonary complications; (5) rapid recovery of intestinal peristalsis; (6) rapid healing of the wound; (7) stronger scar, and incidence of incisional hernia is very low; 1 in 1250 cases; (8) more cosmetic (bikini incision).

Disadvantages

(1) It takes a longer time to open and close the abdomen; (2) there is more bleeding caused by dissecting the anterior rectus sheath from the underlying rectus muscle. In fact the main complication is haematoma formation; (3) less exposure compared with midline incision and does not allow dealing with problems in the upper abdomen. For this reason it is not done when we operate for a malignant lesion in the pelvis.

VERSION

It means changing the lie of the fetus inside the uterus.

TYPES

When the head is made to present it is called cephalic version and when the breech is made to present it is called podalic version. There are 2 types of version:

1. External cephalic version (page 186).
2. Internal podalic version.

INTERNAL PODALIC VERSION

It is only indicated to deliver a retained second fetus in case of twins. However, it can be applied to deliver a small dead fetus in a transverse lie.

Requirements

1. The cervix must be fully dilated.
2. The membranes are intact or recently ruptured so there is enough amount of liquor amnii.
3. Deep general anaesthesia and aseptic technique.

Contraindications

1. Incompletely dilated cervix.
2. Drainage of the liquor amnii.
3. The presence of uterine scar as after caesarean section.

Technique

General anaesthesia. Lithotomy position. Bladder is evacuated by a catheter. Episiotomy is performed in the primigravida. The whole hand is introduced through the cervix into the uterus to grasp one or both feet. The foot is brought down while the head is pushed upwards towards the fundus by the external hand. When version is completed, breech extraction is performed. Finally the uterus and birth canal are explored to detect any tear.

Note: In transverse lie bring down the lower foot first if the back is anterior and the upper foot first if the back is posterior so that the back is kept anterior during delivery as breech.

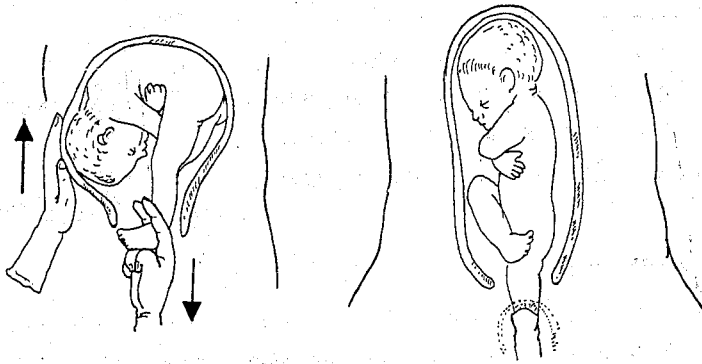


Fig.130. Internal podalic version

Differentiation Between a Foot and a Hand

The foot is known by feeling the heel (the only reliable sign). The toes are short and their tips form a straight line. The hand is known by the freely mobile thumb also the fingers are long and their tips form a curved line. The elbow is also sharper than the knee.

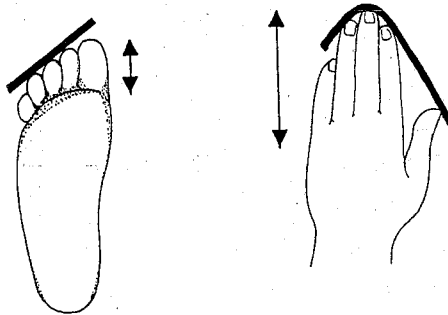


Fig. 131. Differentiation between a foot and a hand

Complications

A) Maternal

1) Rupture of uterus; (2) cervical lacerations and postpartum haemorrhage; (3) puerperal sepsis.

B) Fetal

Complications of breech delivery.

THE OBSTETRIC FORCEPS

TYPES

I. Wrigley Forceps

It is a short curved forceps. The length is 11 inches, the blades, the shanks and handles are short and it is used when the head is low or during caesarean section. The main indication is rigid perineum.

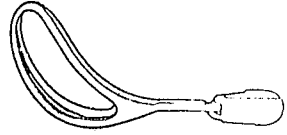


Fig.132. Wrigley forceps

II. Long Curved Forceps

It consists of two blades of stainless steel. The blades are named right and left according to the side of the maternal pelvis into which the blade is introduced. The blade is about 15 inches in length and is made of four parts: the blade proper, shank, lock, and handle.

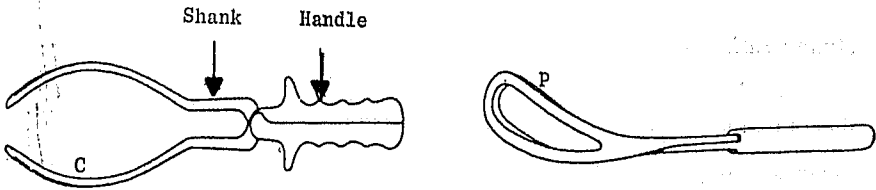


Fig.133. Long curved forceps. (C) Cephalic curve. (P) Pelvic curve



Fig.134. English lock

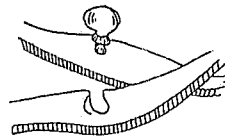
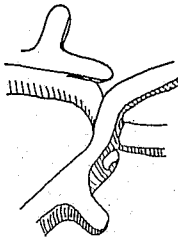


Fig.135. French lock

1. The blade proper: 7 inches in length, fenestrated to be light, to minimize compression of the fetal head and to avoid slipping of the forceps by allowing the parietal eminences to protrude through the fenestra. It has two curves: a cephalic curve and a pelvic curve. The cephalic curve fits over the fetal head. The pelvic curve has 2 borders; anterior border which is concave and must be directed anteriorly towards the symphysis pubis, and a posterior border which is convex and runs parallel with the curve of the sacrum. The advantage of the pelvic curve is to grasp the head by the biparietal diameter to keep the head flexed during traction (central grip of the head). The separation between

the tips of the blades is 1 inch and the separation between the two blades at the centre is 3.5 inches.

2. The shank; connects the handle with the blade proper, about 2 inches in length. It allows locking of the blades outside the vagina and minimizes the force of traction.
3. The lock: To articulate the 2 blades. There are four types:
 - a) The English double slot lock.
 - b) The French screw lock.
 - c) The German lock which is a combination of the above two types.
 - d) The sliding lock, in Kielland forceps.
4. The handle; 6 inches in length. It may be smooth or serrated to avoid slipping. It may have a projecting shoulder for traction.

III. Axis Traction Forceps

It is a long curved forceps to which is attached an axis traction piece. The value of the axis traction piece is to allow traction in the correct direction that is downward and backward, and to avoid loss of force against the symphysis pubis.

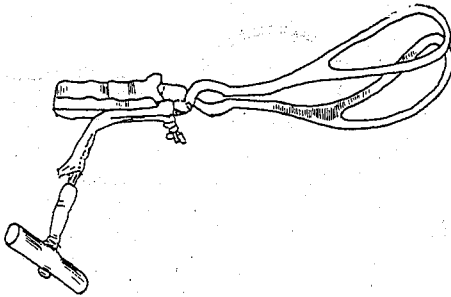


Fig.136. Simpson Neville axis traction forceps

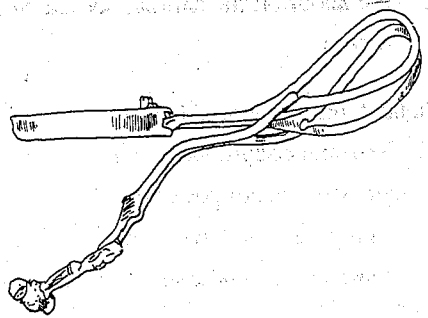


Fig.137. Milne Murray forceps

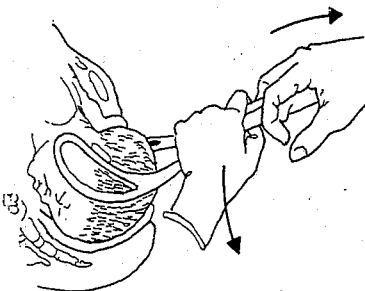


Fig.138. Pajot technique

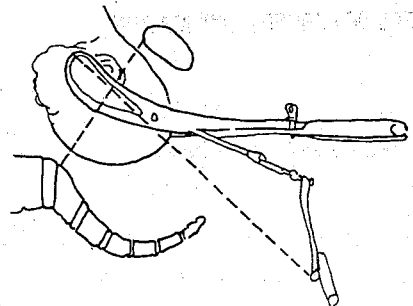


Fig. 139. Traction on the handle assures traction in the proper direction

Types of Axis Traction Forceps

1. The Simpson-Neville axis traction forceps in which the axis traction piece is attached to the handles.
2. Milne-Murray forceps: A traction rod is attached to the blade proper from the outside, and the two rods are connected to an axis traction handle. It is difficult to apply the blade because of the rod but traction is applied directly over the head.

If there is no axis traction piece the Pajot technique is used. The operator applies backward pressure on the shanks by the left hand while pulling downward with the right one on the handles until the head reaches the perineum. The resultant force will be downward and backward.

IV. Kielland Forceps

It is characterized by:

1. A very slight pelvic curve. This allows rotation of the head and its extraction by a single application.
2. A sliding lock. This permits application of the forceps on asynclitic head.
3. The knobs on the handles should be directed towards the occiput when applying the forceps.

Indications of Kielland Forceps

1. Persistent occipito-posterior.
2. Persistent mento-posterior.
3. Deep transverse arrest of the head.

However, in these cases caesarean section is safer for both mother and fetus.

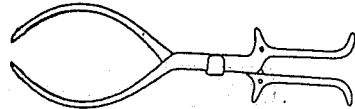


Fig.140. Kielland forceps

FUNCTIONS OF THE FORCEPS

1. Traction; which is the main action.
2. Rotation of the head as in case of deep transverse arrest, persistent occipito-posterior or persistent mento-posterior.

TYPES OF FORCEPS APPLICATION

1. *Cephalic application:* The blades are applied on both sides of the head to grasp the biparietal diameter. This application is safe for the fetus because there is minimal compression of the head.
2. *Pelvic application:* The blades are introduced into the right and left side of the pelvis irrespective of the position of the head. This application is easy but it is dangerous if internal rotation of the head has not occurred so that it will be grasped obliquely.

3. *Cephalopelvic application*: If the occiput is directly anterior or directly posterior the two applications coincide. This is the ideal application because it is easy and safe for the fetus.

TYPES OF FORCEPS OPERATION

1. *High forceps*: This means application of the forceps to a high head, i.e. the biparietal diameter is still above the pelvic brim. This is difficult and dangerous for both mother and fetus and is never done nowadays.
2. *Mid forceps*: The forceps is applied to a mid-head, i.e. the biparietal diameter is below the pelvic brim but it has not passed below the level of the ischial spines. An axis traction forceps must be used or we apply Pajot manoeuvre.
3. *Low (outlet) forceps*: The forceps is applied to a low head, i.e. the biparietal diameter has passed below the level of the ischial spines and internal rotation of the head has occurred. In this case the head is visible on the pelvic floor with the sagittal suture in the antero-posterior diameter of the pelvis. Wrigley's forceps is used. The commonest indication for low forceps is rigid perineum.

Note: Most obstetricians believe that mid forceps like high forceps should not be performed as it can be dangerous for both mother and fetus.

INDICATIONS OF FORCEPS

I. Prolonged Second Stage of Labour

This means that the second stage lasts more than one hour in the primigravida or more than half an hour in the multigravida. The causes of prolonged second stage that indicate delivery by forceps are:

1. Causes in the powers:
 - a) Weak uterine contractions.
 - b) Contraction ring.
 - c) Inability of the patient to bear down.
2. Causes in the passages
 - a) Minor degree of cephalopelvic disproportion.
 - b) Rigid perineum.
3. Causes in the passenger
 - a) Malposition of the head as persistent occipito-posterior, persistent mento-posterior and deep transverse arrest.
 - b) Short cord (rare).



II. Fetal Indications

1. Fetal distress (page 295).
2. Prolapsed pulsating umbilical cord when the cervix is fully dilated and the head is engaged.
3. The aftercoming head in breech delivery (Piper forceps is ideal).
4. Elective forceps in placental insufficiency.
5. Prophylactic forceps in the preterm baby if there is any perineal rigidity to protect the head from excessive compression.

III. Maternal Indications

1. Maternal distress (page 211).
2. Maternal diseases as heart failure, eclampsia, and pneumonia to shorten the second stage.

N.B.:

- *Elective forceps*: The forceps is kept ready from the start and applied if the second stage is prolonged more than 20 minutes.
- *Piper forceps* is a long curved forceps. It has a perineal curve on its long shank.

CONDITIONS TO BE FULFILLED BEFORE FORCEPS APPLICATION

1. Anaesthesia. General anaesthesia, or pudendal nerve block as in heart failure.
2. Aseptic technique.
3. Absence of obstruction to vaginal delivery either fetal as hydrocephalus, or maternal as pelvic tumour.
4. Adequate pelvic outlet.
5. Bladder must be evacuated by a catheter.
6. Contractions of the uterus must be present; otherwise severe atonic postpartum haemorrhage will occur.
7. Dilatation of the cervix should be complete.
8. Engaged head.
9. Favourable position of the head as occipito-anterior, mento-anterior, or face to pubis.
10. Forewaters, i.e., the membranes should be ruptured to avoid slipping, or premature separation of the placenta by pulling on the membranes.

CONTRAINDICATIONS

Absence of the above mentioned requirements.

FORCEPS IN OCCIPITO-ANTERIOR POSITION

1. Lithotomy position.
2. General anaesthesia or pudendal nerve block (as in heart failure).

3. The vulva, perineum and vagina are painted with an antiseptic solution and sterile towels are applied.
4. The bladder is evacuated by a catheter.
5. Vaginal examination is done under anaesthesia to know the position of the head and to be sure that all the conditions necessary for forceps delivery are fulfilled.
6. The left blade is applied at first. It is grasped by the left hand and introduced in the left side of the pelvis guided by the fingers of the right hand.
7. The right blade is grasped by the right hand and introduced in the same way.
8. The two blades are locked with the left one being lower, then explore to be sure that no soft maternal tissues are included between fetal head and blades of forceps.
9. In case of mid forceps, the axis traction piece is applied.
10. Extraction of the head:
 - a) Traction should be gentle using moderate force.
 - b) Traction is intermittent and better done during a uterine contraction.
 - c) After 3 or 4 pulls the blades are unlocked to relieve continuous compression of the head.
 - d) Traction must be in the correct direction; that is downwards and backwards until the occiput appears then the forceps is gradually elevated upwards.
11. An episiotomy is done in the primigravida or if the perineum is going to tear.
12. After delivery of the head, the forceps is removed and the fetus is extracted.
13. The last step is to explore the uterus and birth canal to detect any tear.
14. The fetus is examined for injury.

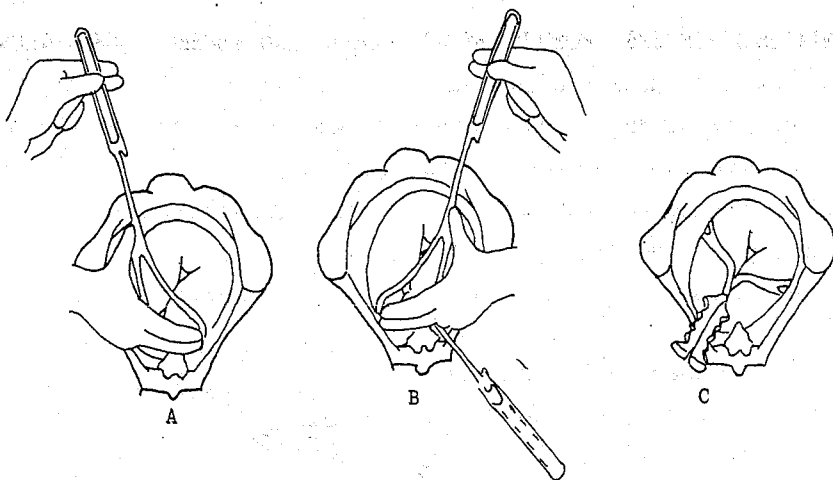


Fig. 141. (A) Application of left blade. (B) Application of right blade (C) Locking of the blades

KIELLAND FORCEPS IN DEEP TRANSVERSE ARREST

The first 5 steps as mentioned before.

1. Orientation. The two blades are locked outside with the knobs on the handles directed towards the occiput to know the anterior and posterior blade.
2. The anterior blade is introduced over the face and is then rotated to lie behind the symphysis pubis. This is called the "wandering technique".
3. The posterior blade is then introduced along the curve of the sacrum.
4. The two blades are locked together. The head is rotated to bring the occiput anteriorly, then traction is applied to deliver the head.
5. After delivery of the head, the forceps is removed, and the fetus is extracted.
6. The uterus and birth canal are explored to detect any tear.
7. The fetus is examined for injury.

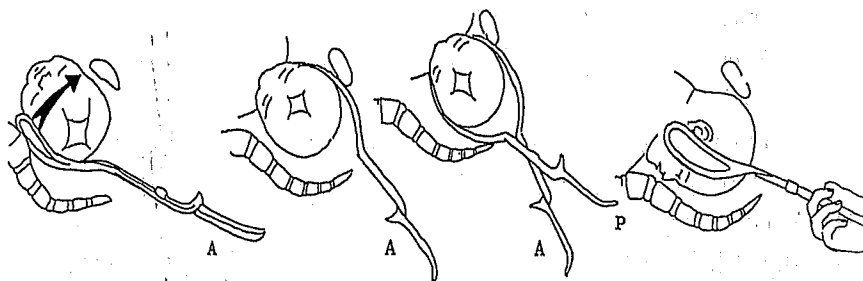


Fig.142. Kielland forceps in deep transverse arrest. (A) Anterior blade
(P) Posterior blade

ADVANTAGES OF FORCEPS APPLIED TO THE AFTERCOMING HEAD IN BREECH PRESENTATION

1. It increases and maintains flexion of the head during its delivery.
2. Traction is applied directly on the head thus avoiding traction on the neck which may lead to brachial plexus injury or fracture of the clavicle.
3. It allows slow delivery of the head, thus avoiding rapid compression which may lead to intracranial haemorrhage.
4. It protects the head from compression by contracted pelvic outlet or rigid perineum.

Piper forceps is the best for the after-coming head because it has a perineal curve on its long shank. If not available the Simpson-Neville forceps is used.



Fig.143. Piper forceps

FAILED FORCEPS

It is failure to apply the blades or to deliver with the forceps.

Causes**I. Maternal**

1. Contracted pelvis.
2. Contraction ring.
3. Generalized tonic contraction of the uterus.
4. Incomplete dilatation of the cervix.
5. Pelvic tumour.
6. Rigid paravaginal tissues.

II. Fetal

1. Large head.
2. Hydrocephalus.
3. Malposition of the head as occipito-posterior, mento-posterior or brow.
4. High head.
5. Large shoulders impacted at the pelvic brim.
6. Double headed monster which is not recognized.

Management

The forceps is removed and the patient is re-examined to know the cause of failure. The treatment is that of the cause.

TRIAL OF FORCEPS

The forceps is applied but keeping in mind that the operation may not succeed. It is mainly indicated in minor cephalopelvic disproportion. If the trial fails, caesarean section is performed, so the procedure must be done in the operating theatre. However, every case of forceps operation should be considered trial of forceps which may fail.

COMPLICATIONS OF FORCEPS DELIVERY**A) Maternal**

1. Perineal, vulval, vaginal and cervical lacerations. The cervical laceration may extend upwards to involve the lower uterine segment (ruptured uterus), anteriorly to involve the bladder (urinary fistula), laterally to involve the uterine vessels or the ureter, posteriorly to open the peritoneum of Douglas pouch leading to peritonitis.
2. Postpartum haemorrhage; traumatic due to laceration of soft tissues or atonic when delivery is performed in absence of uterine contractions.

3. Puerperal sepsis.
4. Pelvic joints injury as separation of the symphysis pubis, dislocation of the sacro-iliac joint or fracture of the coccyx.
5. Pelvic nerves injury as sciatic nerve.
6. Late complications include prolapse, stress incontinence, habitual abortion or habitual preterm labour due to incompetent cervix.

B) Fetal

1. Lacerations of the scalp and face.
2. Cephalhaematoma.
3. Fracture of the skull bones.
4. Intracranial haemorrhage.
5. Injury of facial nerve and facial palsy.
6. Injury of brachial plexus.
7. Injury of the eyes.
8. Injury of the ear and deafness.
9. Asphyxia resulting from cord compression between the head and forceps and from intracranial haemorrhage.

These complications can be avoided if certain conditions are fulfilled before application of forceps (page 362).

Note: The rate of forceps delivery has declined from 5% to 1%.

THE VACUUM EXTRACTOR (VENTOUSE)

It is an instrument used to apply traction on the fetal head after creating a negative pressure or vacuum between its cup and the head. The negative pressure is raised by 0.2 kg/cm² every 2 minutes to a maximum of 0.8 kg/cm². The vacuum is created manually or electrically. The cup is available in 3 sizes (4, 5 and 6 cm in diameter). Soft silastic cups are preferred to metal cups as the soft cup is easier to apply. The largest possible cup (5 or 6) is applied because with a large size, less negative pressure will be applied for cm² over the scalp and this will be less traumatic. The negative pressure creates an artificial caput succedaneum (chignon) which disappears spontaneously after delivery.

INDICATIONS

1. The same indications of the obstetric forceps.
2. Uterine inertia before full dilatation of the cervix (7 cm or more).
3. Prolapsed pulsating cord before full dilatation of the cervix (7 cm or more).

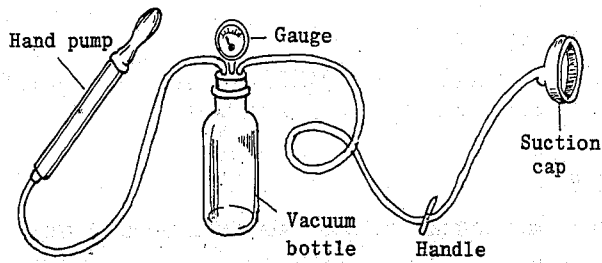


Fig.144. Vacuum extractor

ADVANTAGES OVER THE OBSTETRIC FORCEPS

1) It does not compress the fetal head; (2) less traumatic to maternal tissues; (3) it can be used without anaesthesia so it is ideal for cardiac patients; (4) it can be used before the cervix is fully dilated (7 cm or more); (5) it can be applied to a high head; (6) it can be applied to the unrotated head; (7) it does not occupy space and so it does not interfere with pelvic capacity.

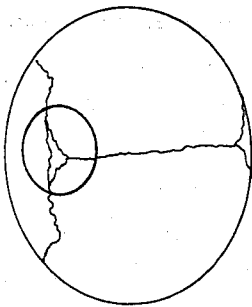


Fig.145. Cup applied near posterior fontanelle to keep head flexed during traction

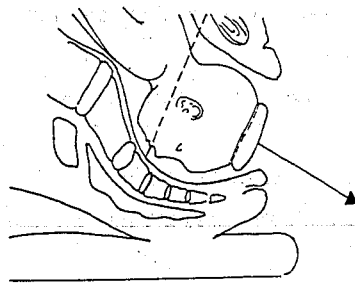


Fig.146. Traction applied perpendicular to the head to avoid slipping of the cup.

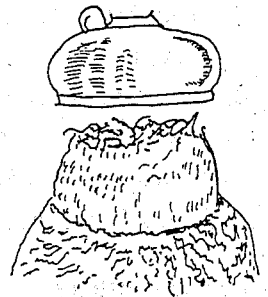


Fig.147. Chignon

COMPLICATIONS

A) Fetal

1. Lacerations of fetal scalp.
2. Cephalhaematoma.
3. Fracture of the skull bones.
4. Intracranial haemorrhage.

B) Maternal

Injury of vagina or cervix if included under the cup.

The cup should not be applied more than 15 minutes to avoid complications. In fact, delivery should be achieved with 5 contractions.

CONTRAINDICATIONS

1) Cephalopelvic disproportion; (2) nonvertex presentation (as face, brow or breech); (3) preterm baby less than 34 weeks for fear of intracranial haemorrhage; (4) high head; this is a relative contraindication.

Note: The rate of ventouse delivery is about 2.5%.

DESTRUCTIVE OPERATIONS ON THE FETUS**I. CRANIOTOMY**

This means perforation of the fetal head and evacuation of its contents to reduce its size.

Indications

1. Hydrocephalus; the commonest indication and the only indication when perforation is performed on a living fetus.
2. When the fetus is dead and there is obstruction to vaginal delivery as in case of:
 - a) Cephalopelvic disproportion.
 - b) Malposition of the head as occipito-posterior, face or brow.
3. Retained aftercoming head in breech and the fetus is dead.

Contraindications

1. If the fetus is alive except in case of hydrocephalus.
2. Marked pelvic contraction with a true conjugate less than 6 cm because the bimaxillary diameter (7.5 cm) is not reduced after crushing of the head.
3. If the cervix is not sufficiently dilated to allow safe introduction of the perforator.

Sites of Perforation

- *Vertex:* Through one parietal bone.
- *Brow:* Through the frontal bone.
- *Face:* Through the orbit.
- *Aftercoming head:* In the occipital bone behind the mastoid process or through the roof of the mouth.

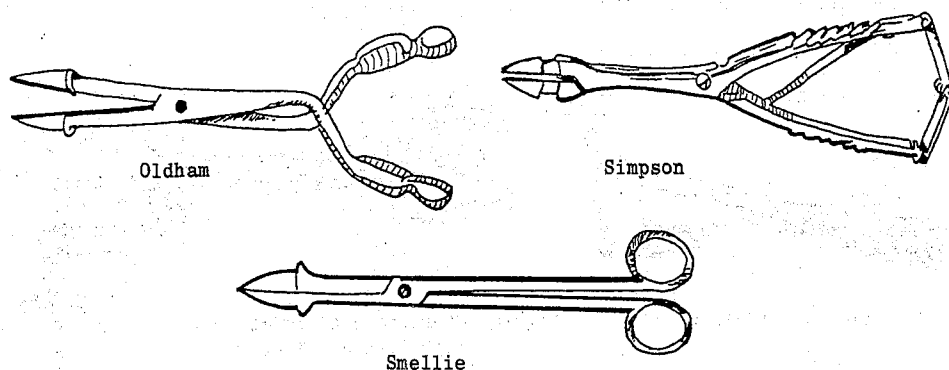


Fig. 148. Perforators

Technique

It is performed under general anaesthesia. The bladder is evacuated by a catheter. Vaginal examination is done under anaesthesia to confirm the diagnosis and to locate the site of perforation. The instrument used is either Oldham or Simpson perforator. The perforator is introduced by one hand and guided by two fingers of the other hand to reach the site of perforation. The perforator is applied perpendicular to the bone to avoid slipping. The perforator is pushed by a rotatory movement through the bone down to its shoulders. The blades are opened to form a linear incision then closed. The perforator is rotated at right angle, and the blades are opened again thus making a cruciate incision in the bone. The perforator is then pushed until the base of the skull to destroy the medulla to ensure that the fetus is dead. A Bozemann catheter was used in the past to wash the cranial contents with sterile water.

Delivery of the Perforated Head

1. Spontaneous delivery; if there is no marked disproportion.
2. By the obstetric forceps; but it is liable to slip.
3. Traction by volsellum or by Willett scalp forceps.
4. By the cranioclast to crush the face. It is now obsolete.
5. By the combined cranioclast and cephalotribe to crush the face and occiput. It is also obsolete.

The last step is to explore the uterus and birth canal to detect any tear.

Complications

1) Infection and puerperal sepsis; (2) perforation of uterus; (3) injury of the soft tissues leading to haemorrhage, and fistula, e.g. vesicovaginal fistula.

Note: Tapping of the hydrocephalic head to reduce its size can be done abdominally or vaginally using a spinal needle or a trocar without anaesthesia (see page 220).

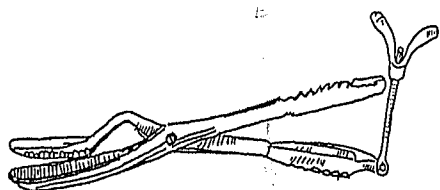


Fig. 149. Cranioclast

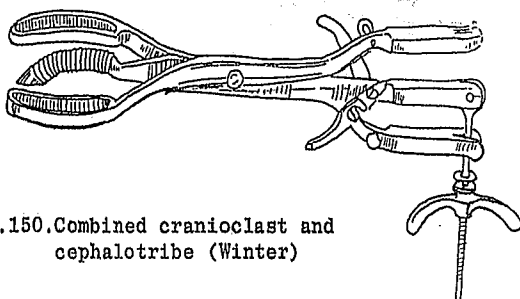


Fig. 150. Combined cranioclast and cephalotribe (Winter)

II. DECAPITATION

It means division of the fetal neck to separate the head from the body. Not done nowadays. Mentioned for those interested in the history of obstetrics.

Indications

1. Neglected shoulder (main indication).
2. Some cases of locked twins.
3. Double headed monster.

Instruments

A) Narrow decapitation hooks

1. Braun decapitation hook.
2. Jardine decapitation hook.

B) Wide decapitation hooks:

1. Ramsbotham decapitation hook with a knife.
2. Galabin decapitation hook which is serrated.

C) Decapitation wire which is safer than the hooks.

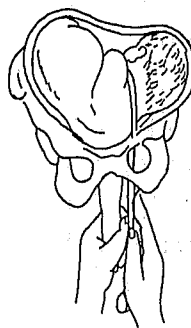


Fig. 151. Decapitation

Technique in Neglected Shoulder

1. General anaesthesia, and the bladder is evacuated by a catheter.
2. Vaginal examination is done under anaesthesia to confirm the diagnosis.
3. If the arm is prolapsed, the assistant pulls it down to make the neck accessible.
4. The decapitation hook is introduced by one hand and guided by two fingers of the other hand. The hook is pushed to reach behind the fetal neck and it is then rotated at right angle to surround the neck.
5. The neck is divided by a sawing upward and downward movement if a sharp hook is used. If a blunt hook is used, it is rotated from side to side to fracture the cervical spine and after removing the hook, the soft tissues of the neck are divided by a long scissors.

6. The trunk is delivered by traction on the arm. Protect the stump of the neck by the hand to avoid injury of maternal tissues by spicules of bone.
7. The head is then delivered by jaw traction helped by suprapubic pressure or with the forceps.
8. The last step is to explore the uterus and birth canal to detect any tear.

Complications: as craniotomy.

Note: Nowadays caesarean section is done because it is safer for the mother than decapitation.

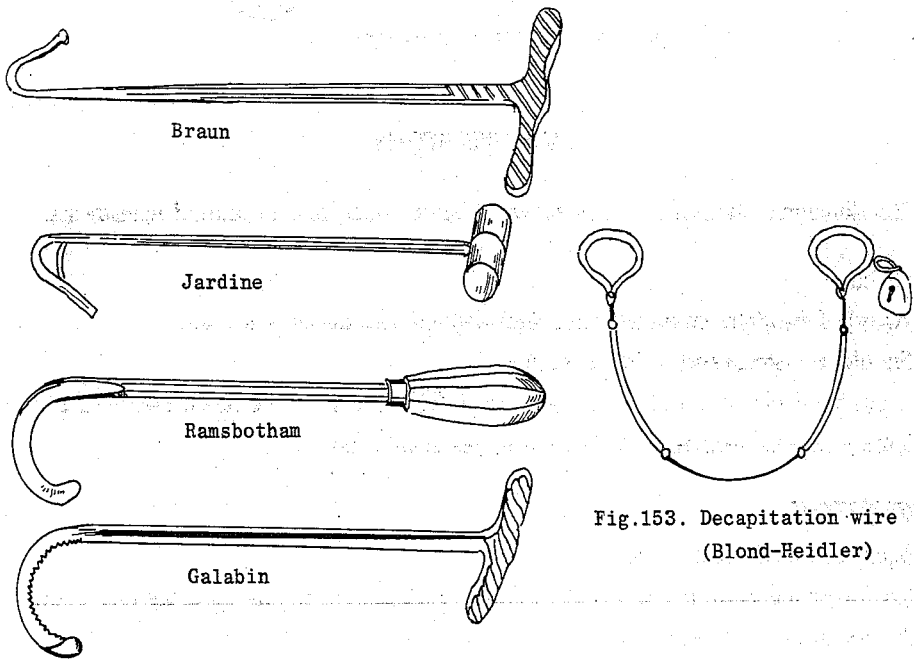


Fig.152. Decapitation hooks

Fig.153. Decapitation wire
(Blond-Heidler)

III. SPONDYLECTOMY

It is division of the spinal column of the dead fetus with a strong scissors. It was indicated in case of neglected shoulder when the neck is out of reach so decapitation cannot be performed. The lower half of the body is delivered first by pulling on the feet and then the upper half by pulling on the arm. Caesarean section is safer.

IV. EVISCERATION

It means opening the abdomen or thorax of the dead fetus and removing the viscera or allowing fluid out to diminish the size of the fetus. The indications are fetal ascites, thoracic and abdominal tumours.

V. CLEIDOTOMY

It is division of one or both clavicles with a strong scissors to reduce the size of the shoulders. It is indicated when there is shoulder dystocia and the fetus is dead.

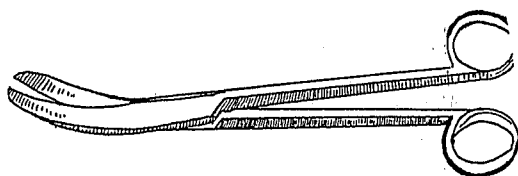


Fig.154. Embryotomy scissors

SYMPHYSIOTOMY

It is division of the symphysis pubis with a knife under local infiltration anaesthesia.

INDICATIONS

1. Arrest of the aftercoming head in breech delivery and the fetus is alive.
2. Shoulder dystocia and the fetus is alive.
3. Contracted pelvic outlet when the forceps fails to deliver the head. Here-symphysiotomy may be done as an alternative to caesarean section.

COMPLICATIONS

1. Injury of bladder and urethra.
2. Haemorrhage from the plexus of veins surrounding the bladder and urethra or from the dorsal vein of the clitoris.
3. Osteomyelitis.
4. Difficulty in walking due to sprain of sacroiliac joints.

EPISIOTOMY

It means incision of the perineum during labour to widen the vaginal orifice.

INDICATIONS

Fetal

- 1) Large head; (2) delivery as face to pubis; (3) breech delivery; (4) preterm baby.

Maternal

1) Primigravida; (2) rigid perineum; (3) after repair of prolapse, complete perineal tear and rectovaginal fistula; (4) if the perineum is overstretched and going to tear; (5) narrow subpubic arch pushing the head posteriorly; (6) intrauterine manipulations or forceps delivery; (7) arrest of the head on pelvic floor more than half an hour; (8) oedema of vulva.

TYPES

1. *Midline episiotomy.* The bleeding is slight and repair is easy and anatomical, less painful in the puerperium and dyspareunia rarely occurs, but it may extend to involve the external anal sphincter and even the rectum.
2. *Medio-lateral episiotomy.* It starts at the midline to avoid injury of Bartholin duct and is directed postero-laterally towards the ischial tuberosity at a 45-degree angle from midline. It avoids damage to the external anal sphincter but there is more bleeding and repair is more difficult and not anatomical, more painful in the puerperium, and more liable to be followed by dyspareunia (tender scar).
3. *J or L-shaped episiotomy.* A midline episiotomy is done. If the wound is going to extend, the incision is carried postero-laterally.

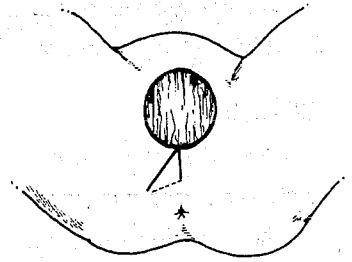


Fig.155. Episiotomy incisions

ADVANTAGES OF EPISIOTOMY

1. It prevents perineal lacerations. The episiotomy wound is easily repaired and heals better than a perineal tear which is irregular.
2. It prevents prolonged or overstretching of the pelvic floor which predisposes to genital prolapse and stress incontinence.
3. It avoids compression of the fetal head and intracranial haemorrhage in case of breech and preterm delivery.
4. It shortens the second stage of labour.

COMPLICATIONS

1. Primary haemorrhage.
2. Haematoma formation. This may be vulval, paravaginal, or in the ischiorectal fossa.
3. Infection of the wound.
4. Breakdown of the episiotomy wound due to infection, bad repair, excessive amount of suture material used leading to ischaemia of tissues, or poor power of healing.

5. Local pain. This may interfere with sitting and if severe it can cause retention of urine due to reflex spasm of the internal urethral sphincter.
6. Extension of the wound may lead to injury of the external anal sphincter and even the rectum. This leads to faecal incontinence.
7. Incomplete healing of a tear involving the rectum leads to a rectovaginal fistula.
8. Injury of Bartholin duct and formation of a Bartholin cyst.
9. Dyspareunia due to the presence of a tender vaginal scar or narrowing of the vagina.
10. Inclusion (implantation) dermoid cyst if part of the skin is included inside the wound.

TECHNIQUE

Local infiltration anesthesia as 1% Lignocaine (10 ml). Episiotomy is done when 3–4 cm of the head appear during a uterine contraction. The incision involves the lower vagina, fourchette, perineal skin and muscles and must be deep to cut the levator ani. Repair is done after delivery of the placenta using chromic catgut or delayed-absorbable suture (as Vicryl or Dexon). The vagina is approximated by continuous or interrupted sutures, then the perineal muscles by continuous or interrupted sutures and finally the skin by interrupted or subcuticular stitches. In fact, there is a great variation in the technique and suture materials used. The wound is kept clean and dry. After each micturition or defecation the perineum is washed with antiseptic solution as Savlon, dried and painted with alcohol and covered by a sterile vulval pad.

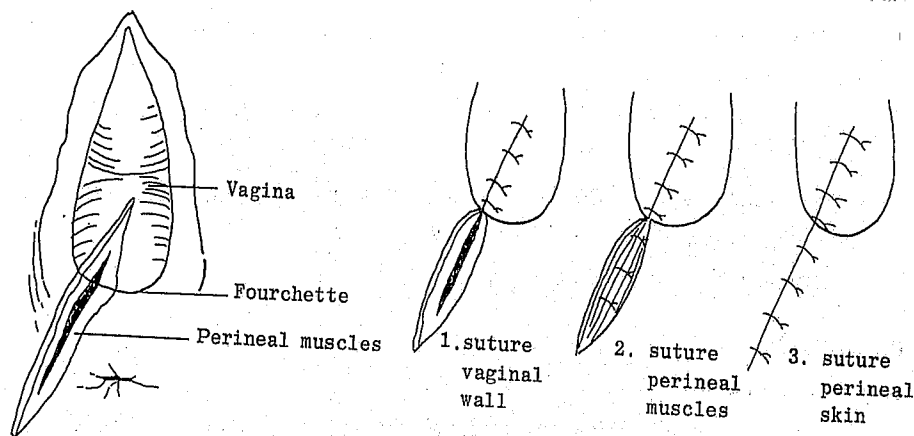


Fig.156. Repair of an episiotomy

OBSTETRIC INSTRUMENTS

Mouth Gag

Used during eclamptic fit to avoid biting of the tongue. It should be covered with rubber (page 84).

Tongue Forceps

Used during eclamptic fit to avoid falling of the tongue backwards and obstruction of the airway (page 84).

Mucus Catheter

To aspirate mucus, meconium and amniotic fluid from the mouth, nose and pharynx of the fetus immediately after delivery. It has a safety chamber where mucus collects. It is now replaced by electric suction (page 146).

Drew Smythe Catheter

Used for rupture of the hindwaters to induce labour and in case of polyhydramnios (page 348).

Doyen Retractor

Used during caesarean section to keep the urinary bladder away from the lower uterine segment (page 353).

Wrigley Forceps

To deliver a low head and during caesarean section if it is difficult to deliver the head manually (page 358).

Long Curved Forceps (page 358).

Simpson-Neville Axis Traction Forceps (page 359).

Milne-Murray Forceps (page 359).

Kielland Forceps (page 360).

Piper Forceps (page 364).

Vacuum Extractor (page 367).

Perforators (page 369).

Cranioclast (page 370).

Combined Cranioclast and Cephalotribe (page 370).

Decapitation Hooks and Decapitation Wire (page 371).

Embryotomy Scissors

For destructive operations performed on a dead fetus as evisceration and cleidotomy (page 372).

Pelvimeters

To measure pelvic diameters.

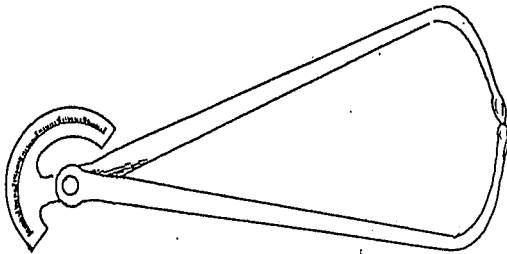


Fig. 157. Martin pelvimeter

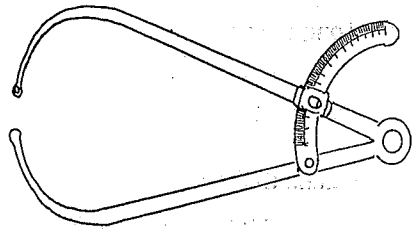


Fig. 158. Crossing pelvimeter (Collins)

Ovum Forceps

To remove products of conception during evacuation.

Ring Forceps

It has a lock on its handles. It is used to:

1. Grasp the soft cervix of the pregnant uterus, e.g. during evacuation or Shirodkar operation.
2. Remove the products of conception during evacuation.
3. Hold the cervix after labour to exclude cervical lacerations.
4. Remove endometrial or cervical polypi.
5. Hold a piece of sponge to remove blood or discharge.



Fig. 159. Ovum forceps

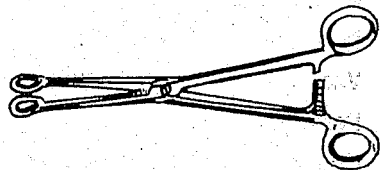


Fig. 160 Ring forceps

Willelt Scalp Forceps

1. To apply traction on the head after craniotomy.

2. To apply traction on the head in certain cases of placenta praevia if there are no facilities for caesarean section.
3. To deliver the head during caesarean section.

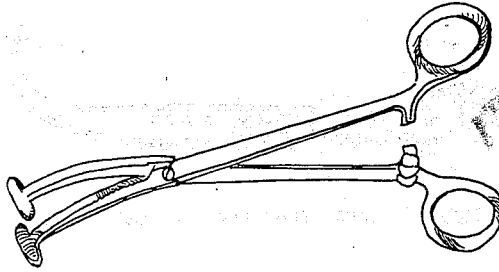


Fig.161. Willett scalp forceps

Bozemann Double Way Catheter

It was used in the past:

1. After craniotomy to wash out the cranial contents.
2. To perform a hot intrauterine douche in atonic postpartum haemorrhage. It is double way to prevent the passage of the fluid along the fallopian tubes which may cause peritonitis.

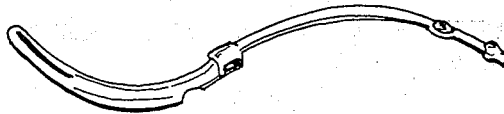


Fig.162. Bozemann double way catheter

Blunt hook

It may be used for groin traction on a dead fetus in breech presentation.

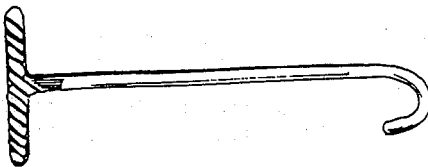


Fig. 163. Blunt hook



Fig.164. Double blunt hook, one end is wider to fit on a large groin

Blunt hook and Crochet

It may be used to deliver a dead fetus in breech presentation. The blunt hook is used for groin traction and the crochet to apply traction on the head after craniotomy.

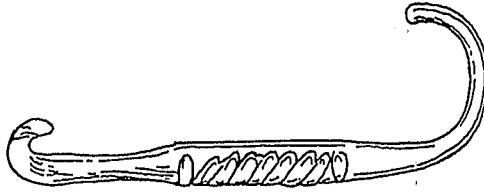


Fig.165. Blunt hook and crochet

Fetal or Pinard Stethoscope

To auscultate fetal heart sounds during pregnancy and labour. It has been replaced by the Doppler stethoscope (Sonicaid).

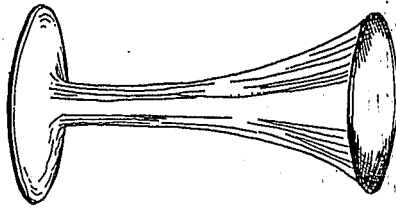


Fig.166. Fetal stethoscope

Green-Armytage Forceps

To hold the edges of the uterine incision during lower segment caesarean section. Also to control bleeding from the edges.

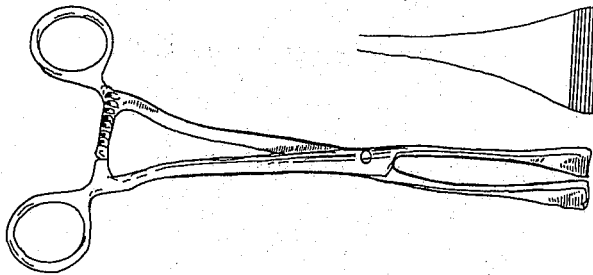


Fig.167. Green Armytage forceps

Aneurysm Needle

Used during Shirodkar operation to pass a silk suture around the internal cervical os.



Fig. 168. Aneurysm needle.

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